“CLINICO – RADIOLOGICAL CORRELATION IN THALAMIC VASCULAR SYNDROMES”

Dissertation submitted to

THE TAMILNADU DR.MGR MEDICAL UNIVERSITY

in partial fulfillment of the requirements

for the award of the degree of

DM (NEUROLOGY) – BRANCH -1

MADRAS MEDICAL COLLEGE

THE TAMILNADU DR.MGR MEDICAL UNIVERSITY

CHENNAI

AUGUST 2014

CERTIFICATE

This is to certify that the dissertation entitled **"CLINICO – RADIOLOGICAL CORRELATION IN THALAMIC VASCULAR SYNDROMES"** is a bonafide record of work done by **Dr. D. SEKAR** in the **Institute of Neurology, Rajiv Gandhi Government General Hospital & MADRAS MEDICAL COLLEGE, CHENNAI** in partial fulfillment of the **Tamilnadu Dr.MGR Medical University** rules and regulations for the award of **D.M. (NEUROLOGY) degree under my direct guidance and supervision during the academic year 2011-2014.**

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ACKNOWLEDGEMENT

It gives me great pleasure to acknowledge all those who guided, encouraged and supported me in the successful completion of my dissertation.

First and foremost, I express my gratitude to, the **Dean Dr. R. VIMALA, MD.,** for having permitted me to carry out this dissertation work at Rajiv Gandhi Government General Hospital, Madras Medical College, Chennai.

I am extremely thankful to **Prof. Dr. K. MAHESHWAR, M.ch.** Professor of Neurosurgery, Head of the department, Institute of Neurology, Rajiv Gandhi Government General Hospital, Chennai for his constant encouragement, valuable guidance and support.

I express my deep sense of gratitude and sincere thanks to our respected and beloved Chief **Prof. Dr. R.LAKSHMI NARASIMHAN, MD., DipNB., D.M., DipNB., Professor of Neurology,** Institute of Neurology, Rajiv Gandhi Government General Hospital, Chennai for his valuable suggestions, constant motivation, kind guidance and moral support without which this study would not have been possible.

I express my sincere thanks and gratitude to our Professors **Prof. Dr. K.Bhanu,Dip.NB.,D.M., Prof. Dr.G.Sarala,MD.,DM., Prof.Dr.M.Balasubramanian,MD.,DM**., **Prof.Dr.V.Kamaraj,MD.,DM.,** for their valuable suggestions and support.

I am extremely thankful to all my **Assistant Professors** for their valuable guidance and support.

I owe my sincere thanks to all the patients and the technical staff who participated in the study for their cooperation which made this study possible.

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INTRODUCTION

Stroke is a major cause of death and disability worldwide and the second most common cause of death. It is a common cause of dependency among all the neurological disorders. [1,2].

According to World Health Organization (WHO) stroke is defined as rapidly developing clinical symptoms and / or signs of focal, at times global loss of cerebral function, with symptoms lasting more than 24 hours or leading to death, with no apparent cause other than of vascular origin.

The global prevalence of stroke is 5 to 8 /1000. Globally stroke incidence varies according to the ethnic differences in a common geographical location and ranged from 93 to 223/1,00,000 population. The incidence of stroke in India is difficult to study due to multiple factors.

The stroke risk increases steadily as the age advances. The term *stroke* is applied to a sudden focal neurologic syndrome,[1] caused by cerebrovascular disease. The vascular pathologic process may be considered not only in its grosser aspects-embolism, thrombosis, dissection, or rupture of a vessel-but also in terms of more basic or primary disorder, i.e., atherosclerosis, hypertensive arteriosclerotic change, arteritis, aneurysmal dilatation, and developmental malformation. Most strokes are sudden onset of a focal

neurologic deficit .Stroke is the one, which is common among many neurological disorders. Worldwide, stroke is one of the leading causes of death and a major etiology for adult disability. Stroke poses serious problems with medical, rehabilitation and socioeconomic status. As the prevalence of disability due to stroke is expected to rise due to increase in population, this burden will still increase over the next few decades.

Stroke is the one of the important causes of long term disability in a community setting as about 30 to 50% of stroke patients are left with residual deficits. The hospital based studies had shown that 2% of all, 4 to 5 % of medical and 20% of neurological admissions were due to stroke.

Strokes occur either in anterior circulation or posterior circulation. Posterior circulation supplies approximately one-fifth of the total brain. These areas include cerebellum, brainstem, occipital lobes, medial temporal lobes and thalamus. Posterior circulation is formed by 1 basilar artery, 2 vertebral arteries and 2 posterior cerebral arteries[3].

Thalamus can also be affected as infarct or haemorrhagic stroke.

Thalamic stroke can occur in isolation or in combination with other areas of involvement. isolated involvement of thalamus is not very common compared to other areas.

Unlike other regions, thalamus has got different regions and multiple different nuclei and connected to different areas of brain through extensive connections with afferent and efferent fibres. It has got different blood supply.

Hence , thalamus subserves different functions ranging from touch sensation to maintaining arousal state.

Hence a thalamic lesion can have different and varied manifestations ranging from a common and classical hemisensory loss or hemiparesis and may cause comatose state.

The risk factors for thalamic vascular syndromes are similar to other areas of stroke. As like other areas of stroke the incidence of thalamic stroke increases in old age.

AIM OF THE STUDY

- To analyze the clinical profile of thalamic stroke patients
- To analyze the radiological profile of thalamic stroke patients
- To correlate the clinical and radiological profile of thalamic stroke patients

REVIEW OF LITERATURE

General Appearances of the Thalamus

The thalamus is a large, egg-shaped mass of graymatter . It forms the major part of the diencephalon. There are two thalami . One is situated on each side of the third ventricle . The anterior end of the thalamus is narrow and rounded and it forms the posterior boundary of the interventricular foramen. The posterior end is expanded to form the pulvinar, and overhangs the superior colliculus . The inferior surface is continuous with the tegmentum of the midbrain. The medial surface of the thalamus forms part of the lateral wall of the third ventricle .It is usually connected to the opposite thalamus by a band of gray matter , the interthalamic connection .

Subdivisions of the Thalamus

The thalamus is covered on its superior surface by a thin layer of white matter, called the stratum zonale , and on its lateral surface by another layer, the external medullary lamina . The gray matter of the thalamus is divided by a vertical sheet of white matter, the internal medullary lamina, into medial and lateral halves . The internal medullary lamina consists of nerve fibers and pass from one thalamic nucleus to another. Anterosuperiorly, the internal medullary lamina splits, resembling a Y shape. The thalamus hence is

subdivided into three main parts; the anterior part lies between the limbs of the Y, and the medial and lateral parts lie on the sides of the stem of the Y .

Each of the three parts of the thalamus contains a group of thalamic nuclei .The smaller nuclear groups are situated within the internal medullary lamina, and some are located on the medial and lateral surfaces of the thalamus.

Anterior Part

The anterior part of the thalamus contains the anterior thalamic nuclei . They receive the mammillothalamic tract from the mammillary nuclei. The anterior thalamic nuclei also receive reciprocal connections with the cingulate gyrus and also hypothalamus. The function of the anterior thalamic nuclei is closely associated with that of the limbic system and is concerned with emotional tone and also the mechanisms of recent memory.

Medial Part

The medial part of the thalamus contains the large dorsomedial nucleus and also several smaller nuclei . The dorsomedial nucleus has two-way connections with the whole prefrontal cortex of the frontal lobe of the cerebral hemisphere. It also has similar connections with the hypothalamic nuclei. It is interconnected with all other groups of thalamic nuclei. The medial part of the thalamus is responsible for the integration of a large variety of sensory

information, including somatic, visceral, and olfactory information, and the relation of this information to one's emotional feelings and subjective states.

Lateral Part

The nuclei are subdivided into a dorsal tier and a ventral tier .

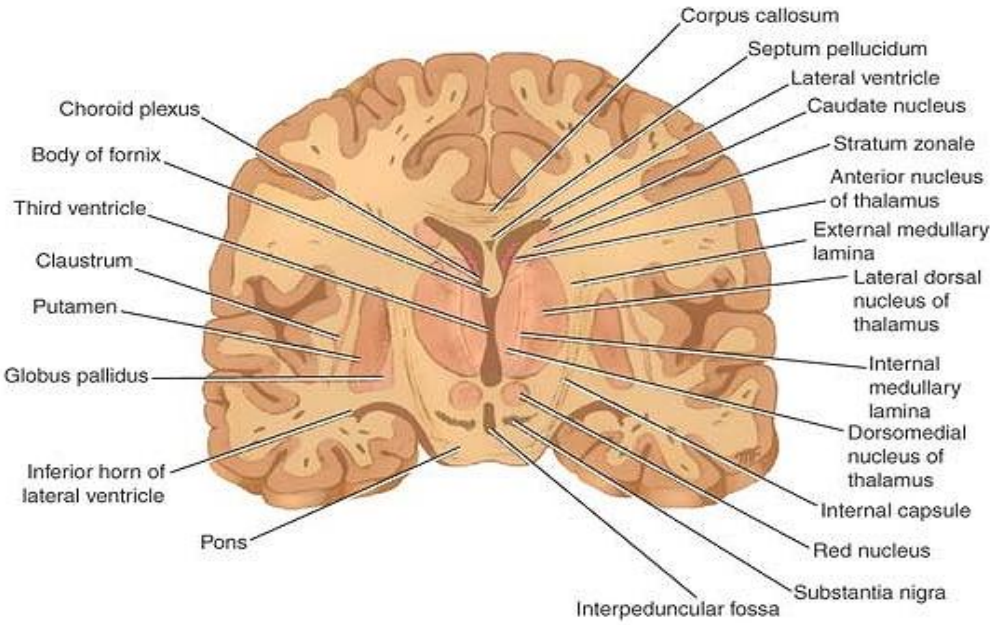
Dorsal Tier of the Nuclei

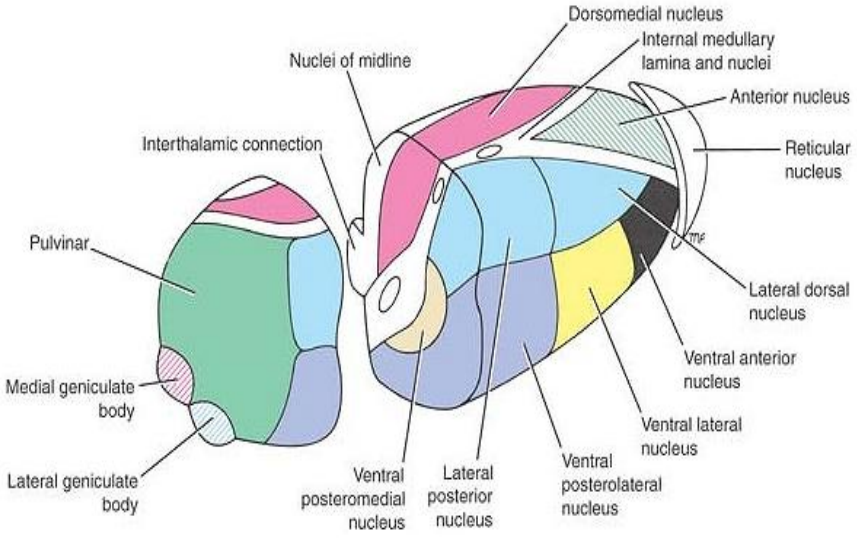
The dorsal tier includes the lateral dorsal nucleus, the lateral posterior nucleus, and the pulvinar. They are known, to have interconnections with other thalamic nuclei and with the parietal lobe, cingulate gyrus, and occipital and temporal lobes.

Ventral Tier of the Nuclei

The ventral tier consists of the following nuclei in a craniocaudal sequence:

- Ventral anterior nucleus . This nucleus is connected to the reticular formation, the substantianigra, the corpus striatum, and the premotor cortex as well as to many of the other thalamic nuclei. Since this nucleus lies on the pathway between the corpus striatum and the motor areas of the frontal cortex, it influences the activities of the motor cortex.


Corpus callosum
Septum pellucidum
Lateral ventricle
Caudate nucleus
Stratum zonale
Anterior nucleus of thalamus
External medullary lamina
Lateral dorsal nucleus of thalamus
Internal medullary lamina
Dorsomedial nucleus of thalamus
Internal capsule
Red nucleus
Substantia nigra
Interpeduncular fossa
Pons
Inferior horn of lateral ventricle
Globus pallidus
Putamen
Claustrum
Third ventricle
Body of fornix
Choroid plexus


Dorsomedial nucleus
Internal medullary lamina and nuclei
Nuclei of midline
Anterior nucleus
Interthalamic connection
Reticular nucleus
Pulvinar
Lateral dorsal nucleus
Ventral anterior nucleus
Medial geniculate body
Ventral lateral nucleus
Lateral geniculate body
Ventral posteromedial nucleus
Lateral posterior nucleus
Ventral posterolateral nucleus

- Ventral lateral nucleus . This nucleus has connections similar to those of the ventral anterior nucleus. It, has a major input from the cerebellum and a minor input from the red nucleus. Its main projections pass to the motor and premotor regions of the cerebral cortex.Thus this thalamic nucleus probably influences motor activity.
- Ventral posterior nucleus. This nucleus is subdivided into the ventral posteromedial nucleus and the ventral posterolateralnucleus . The ventral posteromedial nucleus receives the ascending trigeminal and gustatory pathways, while the ventral posterolateral nucleus receives the important ascending sensory tracts, the medial and spinal lemnisci. The thalamocortical projections from these important nuclei pass through the posterior limb of the internal capsule and corona radiata to the primary somatic sensory areas of the cerebral cortex in the postcentralgyrus.

The intralaminar nuclei are small collections of nerve cells within the internal medullary lamina . They receive afferent fibers from the reticular formation as well as fibers from the spinothalamic and trigeminothalamic tracts; they send efferent fibers to other thalamic nuclei, which in turn project to the cerebral cortex, and fibers to the corpus striatum. The nuclei are believed to influence the levels of consciousness and alertness in an individual.

The midline nuclei consist of groups of nerve cells adjacent to the third ventricle and in the interthalamicconnection . They receive afferent fibers from the reticular formation.

The reticular nucleus is a thin layer of nerve cells sandwiched between the external medullary lamina and the posterior limb of the internal capsule . Afferent fibers converge on this nucleus from the cerebral cortex and the reticular formation, and its output is mainly to other thalamic nuclei. The function of this nucleus may be concerned with a mechanism by which the cerebral cortex regulates thalamic activity.

The medial geniculate body forms part of the auditory pathway and is a bulge on the posterior surface of the thalamus beneath the pulvinar . Afferent fibers to the medial geniculate body form the inferior brachium and come from the inferior colliculus. Hence the inferior colliculus receives the termination of the fibers of the lateral lemniscus. The medial geniculate body receives auditory information from both ears but predominantly from the opposite ear.

The efferent fibers leave the medial geniculate body to form the auditory radiation, which passes to the auditory cortex of the superior temporal gyrus. The lateral geniculate body forms part of the visual pathway and is a swelling on the undersurface of the pulvinar of the thalamus . The nucleus consists of six layers of nerve cells and is the terminus of all but a few fibers of the optic

tract . The fibers are the axons of the ganglion cell layer of the retina and come from the temporal half of the ipsilateral eye and from the nasal half of the contralateral eye, the latter fibers crossing the midline in the optic chiasma. Each lateral geniculate body, therefore, receives visual information from the opposite field of vision.

The efferent fibers leave the lateral geniculate body to form the visual radiation, which passes to the visual cortex of the occipital lobe.

Connections of the Thalamus

The neuronal loops exist between the thalamic nuclei and other areas of the central nervous system are:

- Every thalamic nucleus except the reticular nucleus sends axons to specific parts of the cerebral cortex , and every part of the cerebral cortex sends reciprocal fibers back to the thalamic nuclei. So, the information received by the thalamus is always shared with the cerebral cortex and that the cortex and thalamus can modify each other's activities.
- The thalamus is an important relay station for two sensory-motor axonal loops involving the cerebellum and the basal nuclei:
- (1) the cerebellar-rubro-thalamic-cortical-ponto-cerebellar loop and

- (2) thecorticalstriatal-pallidal-thalamic-cortical loop, both of which are necessary for normal voluntary movement.

Functions of the Thalamus

The thalamus is made up of complicated collections of nerve cells that are centrally placed in the brain and are interconnected.

- A vast amount of sensory information of all types except smell converges on the thalamus and is integrated through the interconnections between the nuclei. The resulting information pattern is distributed to other parts of the central nervous system. That olfactory information is first integrated at a lower level with taste and other sensations and is relayed to the thalamus from the amygdaloid complex and hippocampus through the mammillothalamic tract.
- Anatomically and functionally, the thalamus and the cerebral cortex are closely linked. The fiber connections have been established, and thatfollowing removal of the cortex, the thalamus can appreciate crude sensations. However, the cerebral cortex is required for the interpretation of sensations based on past experiences. If the sensory cortex is destroyed, one can still appreciate the presence of a hot object

in the hand; however, appreciation of the shape, weight, and exact temperature of the object would be impaired.

- The thalamus possesses certain very important nuclei whose connections have been clearly established. These include the ventral posteromedial nucleus, the ventral posterolateral nucleus, the medial geniculate body, and the lateral geniculate body.

Table 1 The Various Thalamic Nuclei, Their Nervous Connections, and Their Functions

Thalamic Nucleus	Afferent Neuronal Loop	Efferent Neuronal Loop	Function
Anterior	Mammillothalamic tract, cingulate gyrus, hypothalamus	Cingulate gyrus, hypothalamus	Emotional tone, mechanisms of recent memory
Dorsomedial	Prefrontal cortex, hypothalamus, other thalamic nuclei	Prefrontal cortex, hypothalamus, other thalamic nuclei	Integration of somatic, visceral, and olfactory information and relation to emotional feelings and subjective states
Lateral dorsal, lateral posterior, pulvinar	Cerebral cortex, other thalamic nuclei	Cerebral cortex, other thalamic nuclei	Unknown
Ventral anterior	Reticular formation, substantianigra, corpus striatum, premotor cortex, other thalamic nuclei	Reticular formation, substantianigra, corpus striatum, premotor cortex, other thalamic nuclei	Influences activity of motor cortex
Ventral lateral	As in ventral anterior nucleus but also major input from cerebellum and minor input from red nucleus		Influences motor activity of motor cortex
Ventral posteromedial (VPM)	Trigeminal lemniscus, gustatory fibers	Primary somatic sensory (areas 3, 1, and 2) cortex	Relays common sensations to consciousness
Ventral posterolateral (VPL)	Medial and spinal lemnisci	Primary somatic sensory (areas 3, 1, and 2) cortex	Relays common sensations to consciousness
Intralaminar	Reticular formation, spinothalamic and	To cerebral cortex via other	Influences levels of consciousness

	trigeminothalamic tracts	thalamic nuclei, corpus striatum	and alertness
Midline	Reticular formation	Unknown	Unknown
Reticular	Cerebral cortex, reticular formation	Other thalamic nuclei	? Cerebral cortex regulates thalamus
Medial geniculate body	Inferior colliculus, lateral lemniscus from both ears but predominantly the contralateral ear	Auditory radiation to superior temporal gyrus	Hearing
Lateral geniculate body	Optic tract	Optic radiation to visual cortex of occipital lobe	Visual information from opposite field of vision

- The ventroanterior and the ventrolateral nuclei of the thalamus form part of the basal nuclei circuit and thus are involved in the performance of voluntary movements. These nuclei receive input from the globuspallidus and send fibers to the prefrontal, supplemental, and premotor areas of the cerebral cortex.
- The large dorsomedial nucleus has extensive connections with the frontal lobe cortex and hypothalamus. This nucleus lies on the pathway that is concerned with subjective feeling states and the personality of the individual.
- The intralaminar nuclei are closely connected with the activities of the reticular formation, and they receive much of their information from It. Their strategic position enables them to control the level of overall

activity of the cerebral cortex. The intralaminar nuclei are thus able to influence the levels of consciousness and alertness in an individual.

Vascular Supply of the Thalamus

Cerebrovascular disease is the most common cause of discrete thalamic pathology resulting in signs and symptoms of localizing value. Infarcts are more common than hemorrhages. Therefore, knowledge of the vascular supply of the thalamic nuclei helps greatly to understand the so-called thalamic syndromes and the localization of thalamic lesions.

The thalamic arteries arise from the posterior communicating arteries and from the perimesencephalic segment of the posterior cerebral arteries [24]. The origin and territory of supply of the various thalamic vessels differ in each person, like when the posterior communicating artery is small or absent, arterial twigs from the posterior cerebral artery supply the thalamic territory that is otherwise supplied by branches of the posterior communicating artery. The segment of the posterior cerebral artery proximal to the ostium of the posterior communicating artery has been termed the basilar communicating artery[24].

Localization of Ischemic Thalamic Lesions

In localizing ischemic lesions of the thalamus, keep in mind.

- The arterial supply for most of the thalamus arises from the vertebrobasilar system, in some cases with a small contribution from the posterior communicating artery[24]. Only the lateral portion and the hilum of the lateral geniculate body are usually fed by the anterior choroidal artery, a branch of the internal carotid artery.
- Except for the lateral geniculate body, the middle cerebral and anterior choroidal arteries do not supply the thalamus to such an extent that thalamic infarction would result from occlusion of these vessels. So, internal capsular dysfunction, may result from the occlusion of thalamic vessels. Rarely, the anterior cerebral artery may contribute to the supply of the posterolateral thalamus through a posterior pericallosal vessel, which anastomoses with the posterolateralchoroidal artery.
- The paramedian thalamic vessels often arise from a single pedicle that originates in one of the basilar communicating arteries . Thus, unilateral posterior cerebral artery occlusions may result in bilateral paramedian thalamic infarcts[24].

The arterial territory responsible for a thalamic ischemic infarct may be inferred from the clinical findings, as follows.

TABLE 18-2 Vascular Supply of the Thalamus

Name of vessel	Origin	Distribution
Polar arteries	Posterior communicating artery	Thalamic nuclei Reticular Ventral anterior Medial (anterior portion)
Paramedianthalamomesencephalic arteries	Basilar communicating artery (portion of posterior cerebral artery proximal to ostium of posterior communicating artery)	Thalamic nuclei Reticular Ventrolateral Medial Midline (paraventricular) Centromedian Other structures Red nucleus (superior-median portion) Interpeduncular nucleus Decussation of the superior cerebellar peduncle Third nerve nucleus
Thalamogeniculate pedicle	Posterior cerebral artery, proximal to geniculate body level	Ventral caudal nuclei
Posteromedial choroidal arteries	Posterior cerebral artery, just distal to ostium of posterior communicating artery	Thalamic nuclei Centromedian Ventral posterior medial Medial geniculate body Pulvinar Medial (posterior portion) Anterior Other structures Crus cerebri

		Subthalamic nucleus Substantianigra Red nucleus (lateral)
Posterolateralchoroidal arteries	Posterior cerebral artery (between lateral geniculate body and dorsal pulvinar level)	Thalamic nuclei Lateral geniculate body Pulvinar (inferolateral portion) Laterodorsal Other structures Hippocampus Choroid plexus

Paramedian Territory

Infarcts here tend to also involve the paramedian region of the midbrain. Mostly, the syndrome is composed of the clinical triad of somnolent apathy, memory loss, and abnormalities of vertical gaze. Bilateral medial thalamic infarcts account for the behavioral syndrome, and lesions in the area of the rostral interstitial nucleus of the medial longitudinal fasciculus account for the vertical gaze palsy. The following findings may result, depending on the extent and location of the lesion [10, 14, 24] :

- Transient loss of consciousness or somnolence; occasionally akineticmutism

- Behavioral changes (confusion, agitation, aggression, lack of initiative, disorientation, apathy, manic delirium, a frontal lobe-like syndrome[12])
- Recent memory loss (with anterograde and retrograde components); persistent memory loss is observed only with damage of the dominant anterior nucleus or mamillothalamic tract.
- Vertical gaze and convergence disorders (and occasionally blepharospasm)
- Contralateral hemiataxia, asterixis, or motor weakness
- Delayed action tremor (occasionally myoclonus or athetosis) in the contralateral limbs

This syndrome is often due to embolic occlusion of the top of the basilar artery or local atheroma at the origin of the posterior cerebral artery [14, 19].

Thalamogeniculate (Lateral Thalamic or Inferolateral Thalamic) Territory

Ischemia in this territory (ventral posterior nucleus, ventral lateral nucleus, and subthalamic region) causes some of the components of the classic thalamic syndrome described by Dejerine and Roussy [14, 20]:

- Hemianesthesia (occasionally, proprioception is spared)
- Transient slight hemiparesis
- Hemiataxia

- Hemiataxia-hypesthesia syndrome
- Lack of nonvolitional utilization of the contralateral body
- Dysequilibrium
- Choreoathetoid movements
- Athetoid posture
- Paroxysmal thalamic pain
- Homonymous hemianopia mostly due to simultaneous medial occipital infarction

All the findings occur on the contralateral side to the lesioned thalamus. The more severe forms of the syndrome such as complete geniculothalamic infarct accompany proximal occlusion of the posterior cerebral artery. Partial forms (partial geniculothalamic infarct) result from lacunar infarction restricted to one of the penetrating thalamogeniculate vessels [20] and result in pure sensory or sensorimotor stroke. Disease of such small perforating arteries often accompanies diabetes and chronic hypertension.

Isolated hemiataxia and ipsilateral sensory loss (the hemiataxia-hypesthesia syndrome or thalamic ataxia syndrome) may occur with infarction in the thalamogeniculate territory involveing the lateral part of the thalamus such as ventral posterior nucleus and ventral lateral nucleus. The sensory disturbance may be purely subjective, may affect light touch, pain, and temperature sense,

or affect light touch, pain, temperature, position, and vibration sense. The contralateral cerebellar dysfunction and sensory loss is due to a lesion of the dentatorubrothalamic and ascending sensory pathways into the thalamus. Recurrent pure sensory transient ischemic attacks (transient hemihypesthesia) may occur with ventroposterolateral nucleus ischemia.

Tuberothalamic (Anterolateral Thalamic) Territory

Infarcts in this territory are due to thalamopolar artery lesions and result primarily in neuropsychologic dysfunction . The following findings may result:

- Apathy and verbal perseveration, as part of executive function impairment
- Anterograde memory loss
- Facial paresis for emotional movement
- Occasionally, hemiparesis and visual field defects
- The superimposition of temporally unrelated information
- Dysphasia with left-sided lesions
- Hemineglect and impaired visuospatial processing with right-sided lesions

Bilateral polar artery thalamic infarcts result in apathy, abulia,frontal lobe deficits, lethargy, and impaired memory.

Territory of the Posterior Choroidal Arteries

These vessels supply the lateral geniculate body, pulvinar, posterior thalamus, and a small posterior portion of the hippocampus and parahippocampalgyri[13]. In lateral posterior choroidal artery territory infarction, the most common clinical manifestations are:

- Homonymous quadrantanopsia, superior or inferior, or, rarely but particularly suggestive of involvement of the lateral geniculate body in this territory, a homonymous horizontal sectoranopsia, tubular or shaped like a wedge
- Decreased optokineticnystagmus when moving the drum to the side of the lesion
- Hemisensory loss with mild hemiparesis
- Mild hemiparesis, accompanied by sensory loss
- Transcortical aphasia

Isolated medial posterior choroidal artery territory infarction has not been reliably documented.

Miosis, occasionally ipsilateral, has been described with these lesions[13].

Clinical Manifestations of Lesions in the Thalamus

- Since the thalamus is small, several of the nuclei and even several of the functional regions are usually affected simultaneously, even by discrete lesions such as infarcts. Because arteriolar vascular territories cross the nuclear boundaries, ischemic disease affects several nuclei, often partially[24]. Also, many lesions are not restricted to the thalamus, but involve neighboring areas of the brain as well such as, paramedian thalamic vascular lesions tend to affect also the midbrain as well, with a resultant decrease in the level of alertness to the point of coma[24].Hence, other motor or sensory findings that would point to thalamic involvement cannot be elicited. Laterally located lesions may disrupt the internal capsule, thereby causing motor and sensory deficits that mask the deficits characteristically present with thalamic involvement.Hence, the tendency to avoid using an otherwise strong limb contralateral to a affected ventral lateral thalamic area i.e thalamic neglect is not manifest if capsular involvement has resulted in a hemiparesis. Lesions extending inferiorly may yield hemiballismus, for which the subthalamic lesion is primarily responsible. Lesions in the territory of the lateral posterior choroidal artery may cause memory loss through the involvement of the parahippocampalgyrus[13].

- Except for sensory deficits, unilateral thalamic lesions result in transient deficits. But, bilateral lesions or unilateral lesions, such as hemorrhages or tumors, which press against the contralateral thalamus or impinge on the midbrain, may render the patient comatose or akinetic and mute.
- Timing has a particular impact on the clinical expression of thalamic lesions. Once effects of an acute lesion recede, neglect may disappear, inability to walk may yield to mild ataxia, and hemisensory loss diminishes.

Discrete lesions in various regions of the thalamus, such as, deep brain stimulation (DBS) are increasingly used for the treatment of parkinsonian and essential tremor, dystonia, pain[35], epilepsy[17], and the manifestations of Gilles de la Tourette's syndrome[17]. Treatment of the tremor is the most extensively used and best understood DBS thalamic procedure. Essential tremor can be treated by DBS with electrodes in the ventrolateral nucleus. The ventrolateral nucleus includes the nuclei VentralisIntermedius (Vim) and ventralisoralis posterior (Vop). The ideal location of the stimulating electrodes seems to lie in the Vop nucleus immediately anterior to the cerebellar receiving area, Vim.

Disturbances of Alertness

Sudden bilateral paramedian thalamic lesions, such as infarcts, will cause a decreased level of alertness ranging from somnolence to coma [24],usually transient. Prolonged coma may result if the lesion extends into the midbrain tegmentum. These patients often have oculomotorparesis[24]. But, patients with pure thalamic involvement have very small reactive pupils i.e.diencephalic pupil, and their extraocular movements, elicited by the doll's head maneuver, are full[14]. Akineticmutism, may follow bilateral paramedian thalamic lesions[24]

The intralaminar, reticular, and ventral anterior nuclei seem to play the greatest role in mediating normal alertness[17]. Lesions in the region of the intralaminar nuclei cause drowsiness[7]. Electrical stimulation of this region induces arousal from sleep[19]. Bilateral paramedian thalamic infarction may cause severe apathy and a bromocriptine-responsive compulsive tendency assuming a sleeping posture [25]. In patients with paramedian thalamic lesions and daytime hypersomia, rapid eye movement (REM) sleep is normal, but wakefulness, sleep spindling, and stages of deep sleep are all reduced, suggesting that the medial thalamus is the final common pathway, for both maintenance of wakefulness and promotion of non-REM sleep[7, 16]. Low-frequency (3/s), high-intensity combined stimulation of the right centromedian

nucleus and left nonspecific mesencephalic ascending pathways elicited a response similar to the typical absence attack[18]. The dorsomedial thalamic nucleus can be atrophic and hypometabolic on the side of chronic temporal lobe epilepsy[38].

A prion disease mostly confined to the anterior ventral and dorsomedial nuclei has been described fatal familial insomnia in which progressive insomnia with loss of slow-wave sleep and abnormal REM sleep behavior with lack of vegetative and endocrine circadian rhythms and dysautonomia features such as hyperhidrosis, hyperthermia, tachycardia, hypertension, miosis, sphincter disturbances were associated with impaired arousal during daytime, dreamlike states, motor abnormalities such as dysarthria, ataxia, pyramidal dysfunction, intention tremor, myoclonus, and eventual coma and death [27, 36,]. The age of onset of the disease varies between 37 and 64 years. The disease has been related to a mutation at codon 178, or 200 in the prion protein gene (PRNP).

Autonomic Disturbances

DBS with electrodes in the centromedian-DM thalamic region caused a change in penile erection .In these cases, DBS improved the tics of Gilles de la Tourette syndrome. Kleine-Levin syndrome, which is characterized by episodes of somnolence, hyperphagia, impaired recent memory, and hypersexual

behavior, believed to be related to hypothalamic disease, may be due to paramedian thalamic lesions[23].

Disturbances of Mood and Affect

Apathy, disinterest, and lack of drive for motor expression have been reported with lesions of the paramedian region of the thalamus [24,]. Less often, such lesions may cause agitation, dysphoria, or an acute confusionalstate[23], and even undue joviality, accompanied by confabulation[24]. Similar manifestations of bilateral medial thalamic damage may be interpreted as a partial kluver-Bucysyndrome syndrome, in a patient with chronic amnesia, distractibility, hyperorality, affective dyscontrol, and a socially inappropriate behavior[30]. A maniac-like state with disinhibition affecting speech (with logorrhea, delirium, joking, laughing, inappropriate comments, and confabulation) has been described with right thalamic lesions[29]. The ipsilateral cingulate gyrus was hypometabolic. In schizophrenia, a disorder with altered affect and executive function, neuronal loss has been found in the dorsomedial and anterior nuclei of the thalamus [38],

Memory Disturbances

Recent memory may be transiently or permanently impaired by lesions of the anterior or medial thalamic nuclear region . This deficit appears most

consistently with bilateral lesions but may be associated with even unilateral lesions of either thalamus [31].

The proposed anatomic basis for a permanent amnestic syndrome after bilateral anterior thalamic infarctions is combined damage to hippocampal-thalamic pathways through the mammillothalamic tract and medial temporal-thalamic pathways through the inferior thalamic pedicle . These pathways are closely adjacent in the anterior thalamus, and bilateral lesions that cause amnesia are found in this region, whereas bilateral medial thalamic lesions that do not cause amnesia are located more posteriorly .Korsakoff's amnesia correlates with neuronal loss in the anterior thalamic, Pure amnesia has also been described after a unilateral left polar thalamic infarct affecting the anterior thalamic nuclei and adjacent mammillothalamic tract[31]. Lesions involving the left thalamus affect mainly verbal memory, whereas those in the nondominantparamedian thalamic region impair memory related to visuospatial tasks (nonverbal memory) .

Thalamic amnesia is characterized by deficits in anterograde verbal and visual learning and by retrograde amnesia . Motor learning is preserved. Patients who are alert and active usually perform adequately in tests of immediate memory, such as digit span. The amnesia is most profound for events taking place after the injury i.e.anterograde amnesia or recent memory loss, although

it includes information acquired days to years prior to the injury i.e.retrograde amnesia [45]. Disorientation to time is common. With thalamic lesions, the patients may retrieve facts, but in a disorganized fashion and out of context. Some patients seem to be aware of their deficit and others do not.

Unusual patterns of memory loss and recovery have been described with thalamic lesions. A patient with bilateral medial thalamic lesions may recognize the voices of the relatives whom he had failed to identify visually i.e.prosopagnosia.

Confabulation, or falsification of memory occurring in clear consciousness, is frequently present with thalamic amnesia [45]. Patients may confabulate spontaneously or when asked to recall some facts. Particularly those who confabulate spontaneously seem to have an impaired ability to order in time facts retained in memory.

Sensory Disturbances

Thalamic lesions may cause sensory loss, often accompanied by paresthesias and pain.

Paresthesias and Pain

Clinically, small lesions in the ventral posterior lateral nucleus of the thalamus may yield only contralateral paresthesias that lack objective sensory loss when

tested at the bedside[48]. Such paresthesias tend to occur on one side of the face, particularly around the mouth, and in the distal portion of the limbs. Rarely, this cheiro-oral type or distal distribution of the paresthesias may suggest a more distal lesion such as radiculopathy. These areas of the body have the largest representation in the thalamic sensory nuclei. When the trunk is also numb, the subjective feeling of numbness may stop abruptly in the midline, although on objective testing the sensory loss often fades toward the midline. Such a thalamic midline split,□ which is absent with parietal lesions, has been thought to have some clinical value in identifying the site of the lesion[26]. The numb areas of the body may feel swollen, enlarged, shortened, twisted, or torn, or they may tingle. Objects held with the limb contralateral to the lesion may feel abnormally heavy. Lastly, the patient may be unaware of a profound sensory loss.

Pain referred to as thalamic pain is the best known component of Dejerine and Roussy thalamic syndrome[38]. The unpleasant or excruciatingly painful sensation on the side of the body contralateral to a thalamic lesion usually due to infarct may appear at the time of the injury or when the sensory loss begins to improve. The pain feels localized to the skin. Cutaneous stimuli trigger paroxysmal exacerbations of the pain, which persists after the stimulus has been removed. The latency between the stimulus and pain perception is

prolonged, suggesting that the pathways conveying it are polysynaptic. Since the perception of epicritic pain, such as that induced with a pin-prick, is reduced on the painful areas, this symptom has been termed anesthesia dolorosa, or painful anesthesia. Ventroposterior thalamic nuclear lesions are more likely to produce half-body pain than lesions somewhere in the sensory pathways[16]. Some patients with chronic localized neuropatic pain can be relieved by stimulation of the basal ventroposteromedial region of the thalamus. Metabolic studies in such a patient showed that pain relief correlated with increased metabolic rates such as in prefrontal and anterior insular cortices, hypothalamus, and periaqueductal gray, all of them structures thought to play an important role in pain perception.

Thalamic pain seldom occurs with tumors. It has been described most often with vascular lesions, some of which involve not only the thalamus but also the deep parietal white matter[2]. Besides, delayed pain may follow cortical parietal infarcts, particularly those in the bank of the Sylvian fissure, affecting the second somatosensory area i.e.pseudothalamic syndrome .

Loss of Sensory Modalities

All somatosensory modalities are processed in the ventral posterior nucleus of the thalamus contralateral to the side of the body where they are perceived. Within the nucleus there is a topographic distribution: the head is represented anteroinferomedially, the leg is represented posterosuperolaterally; the arm is represented in an intermediate position. A larger volume of the nucleus is for the mouth, tongue, and distal portion of the extremities; their thalamic representation is almost completely crossed. The large oral thalamic and cortical representation in humans may be related to language functions[26]. The face, proximal portion of the limbs, and trunk are represented in a smaller volume of thalamic tissue, mainly contralateral but partially ipsilateral[17]. Thalamic sensory loss tends to occur maximally in the distal portion of the limbs and often spares the face. Such sparing may be related to the different vascular supply of this portion of the ventroposterior region (paramedian territory) or to the bilateral thalamic representation of the face.

Cells concerned with deep pressure and movements of the limbs are preferentially located in the rostral and caudal ends of the ventral posterior lateral nucleus. The central part of the nucleus contains neurons that respond to cutaneous stimuli. In humans, lesions large enough to produce any sensory

loss most often involve several modalities. Pain sensation has been obtained by stimulation of the basal part of the nucleus[17].

Because the perception of pin-prick, temperature, touch, or vibration is altered more often after thalamic than after cortical lesions, these sensory modalities have been termed primary or thalamic. But, conscious joint position identification, two-point discrimination, stereognosis, and graphesthesia tend to be more impaired after cortical parietal lesions, and hence termed secondary or cortical sensory modalities. So, parietal lesions often cause some impairment of thalamic modalities and vice versa. Occasionally, a lesion in the thalamus may disturb mainly the cortical sensory modalities.

Anesthesia and impaired temperature perception tend to occur with basal lesions near the medial geniculate body[16, 17]. The hemispheric lesions causing loss of vibratory sense necessarily implicate the thalamus or the thalamocortical projections.

Decreased thalamic perfusion has been observed with hysterical hemisensory loss. This abnormality reverted when the patients improved.

Disturbances of vision.

Visual field defects caused by thalamic lesions frequently involve the superior quadrant bilaterally. An intolerance to light has been ascribed to a thalamic

lesion[42]. Paramedian thalamic infarction may cause the sudden onset of vivid, formed visual hallucinations i.e. peduncularhallucinosis associated with agitation and sleep disturbanc. Vivid visual hallucinations, suggesting peduncularhallucinosis, with left hemiparesis and left paresthesias is described with a right posterior thalamic infarct. Auditory and visual experiential hallucinations may occur with unilateral thalamic lesions affecting the intralaminar and dorsomedial nuclei. Auditory illusions of hyperacusis and palinacousis may occur with a lesion in the medial geniculate body. Unilateral visual sensory neglect may occur with lesions of the right pulvinar.

A disturbance of olfactory and gustatory perception was noted in a patient with bilateral dorsomedial and intralaminar thalamic lesions. Odors and taste were perceived either in a neutral way, their pleasant character having disappeared, or as unpleasant. Taste sensations have been elicited in humans by stimulation of a portion of the ventroposteromedial nucleus.

Motor Disturbances

Motor disturbances can be related to lesions of the ventral lateral nucleus and the adjacent subthalamic region.

Postural Disturbances

Following an acute thalamic lesion, even a unilateral lesion, patients may be transiently unable to stand or even sit, despite normal strength of the limbs when tested against resistance I e.thalamicastasia. The lesions such as infarction, hemorrhage, or tumor, primarily involved the superoposterolateral thalamus and spared the rubral region. Patients with thalamic astasia cannot stand, and often cannot even sit up unassisted. They fall backward or toward the side contralateral to the lesion and appear to have a deficit of overlearned motor activity of an axial and postural nature. Sudden falling to one side while sitting, standing, or walking has also been described with basal ganglia lesions contralateral to the side of the fall. Some patients with posterolateral thalamic lesions push actively to the side contralateral to the lesion i.e.pusher syndrome. The postural disturbance of thalamic lesions may be accompanied by a disturbance in the patient's perception of the vertical axis .

Postural abnormalities in patients with lesions in the ventrolateral nucleus or its connections with the medial frontal region, in the suprathalamic white matter, ismorethan simple disequilibrium. Volitional movements, are normal. The patient does not use the same strong limbs, and particularly the axial muscles, in tasks that are normally performed automatically such as shifting in bed. Proximal movements that normally support distal ones, are restricted,

even though the patient is perfectly able to abduct his or her shoulder on command. This syndrome occurs with lesions of either thalamus and is different from the sensory hemineglect described predominantly with nondominant thalamic lesions. It has been called motor neglect. Neglect to use the limbs contralateral to the lesion may convey to the examiner the false impression that the patient is hemiplegic . Certain large infarcts, lacunes, hematomas, and tumors may involve the neighboring internal capsule, causing a more or less profound hemiplegia. But, purely thalamic involvement does not result in hemiparesis. Lesions in the ventral lateral nucleus of the thalamus cause contralateral hypotonia, reduction of emotional expression, and transient neglect. This syndrome may occur even with lesions which are discrete enough to spare all sensory modalities and the early components of the somatosensory evoked response. Since the late components of the somatosensory evoked response are abolished, it is postulated that the ventral lateral nucleus plays a key role in the activation of the frontal cortex. Unilateral thalamic lesions cause akinesia, either as a result of sensory inattention or as a consequence of impaired activation of axial, automatic synergies.

Other Motor Disturbances

Lesions of the thalamus may cause emotional facial paresis i.e., weakness of emotionally evoked facial movements, such as smiling, with normal volitional

activation. Contralateral emotional facial paresis has been described with lesions of the thalamus and subthalamus, anterolateral thalamus and insula, posterior thalamus and operculum, and posterior thalamus .

Damage to the dentatorubrothalamic projection to the ventral lateral nucleus by a lesion rostral to the decussation of the superior cerebellar peduncle or damage to the ventral lateral nucleus results in hemiataxiai.e.coarse, with action tremor, dysmetria, dysdiadochokinesia, and rebound of the contralateral limbs. Isolated hemiataxia and ipsilateral sensory loss (the hemiataxia-hypesthesia syndrome) may be a manifestation of thalamic infarction in the thalamogeniculate territory causing damage to the ventral posterior nucleus and ventral lateral nucleus. The cerebellar syndrome is not as severe as with the involvement of the superior cerebellar peduncle or dentate nucleus. Regarding hand movements, pinching may be involved in both cases, but reaching tends to be spared with thalamic lesions[8]. Few weeks after the injury , tremor at a rate of between 3 and 5 cycles per second may appear in the affected extremities. It is mainly distal and increases greatly during the performance of any movement. This tremor may be abolished by stimulation or by a surgical lesion of the ventral lateral nucleus of the thalamus. If the central tegmental tract is also involved, a tremor with a similar rate may affect the eyelids, eyes, or palate i.e.palatal myoclonus or tremor. Such ischemic

lesions occupy the territory of the thalamopeduncularparamedian vessels and are often related to occlusion of the top of the basilar artery[24]. Contralateral cerebellar ataxia and proprioceptive sensory loss may occur with lesions of the ventroposterior thalamus, usually due to interruption of cerebellar outflow pathways in the thalamus rather than to sensory deafferentation.

Lesions that are slightly more rostral, involving the subthalamic region and the pallidothalamic projections to the ventral lateral nucleus, may cause transient contralateral hemiballismus. After few days or weeks, the amplitude of the movement decreases and either disappears or adopts a choreic or athetotic pattern.

Dystonia may be secondary to a necrotizing lesion or to degeneration, as with the familial dystonias. Messenger ribonuclenic acid (RNA) for torsinA, a protein encoded by the gene abnormal in early onset torsion dystonia, is abundantly present in the thalamus[4]. Thalamic infarcts in the intermediate and caudal portions of the ventrolateral nucleus may cause myoclonic dystonia in the contralateral limbs . The myoclonic nature of the deficit, often accompanied by action tremor or chorea, differs from the dystonia with tonic spasms more characteristic of striatopallidal lesions. The onset of the dystonic movements often lags by months or years after the acute insult. Dystonic tremor with chronic MRI evidence of infarction in the anterior nucleus of the thalamus may

have resulted from a more posteroventral lesion causing atrophy but no cavitary lesions in the involved nuclei. The sensory fields of thalamic neurons is enlarged in patients with dystonia. Patients with secondary dystonia or hemiballismus of basal ganglionic origin tend to improve with lesions or stimulation of the ventrolateral nucleus of the thalamus[22].

Abnormal posturing of the hand, often termed thalamic hand, may appear 2 or more weeks after the occurrence of a vascular lesion of the same region. The hand assumes a posture with flexion at the wrist and metacarpophalangeal joints, whereas the interphalangeal joints are hyperextended. Flexion of the metacarpophalangeal joints increases from the second digit, which may actually be extended, to the fifth digit, which is markedly flexed. The fingers may be forcibly abducted. The thumb is either abducted or pushed against the palm.

Other abnormal movements described with thalamic lesions include action myoclonus[5], ideomotor apraxia, and asterixis . Asterixis is more common with ventrolateral nucleus lesions. Hyperekplexia, the sudden loss of postural tone caused by startling stimuli, may be exacerbated with thalamic lesions . Imitation synkineses, also called mirror movements, are common after thalamic lesions .

A decreased corneal reflex may be present in patients with hemiparesis and hemisensory loss due to a cerebral hemispheric lesion. Loss of parietal excitatory influence on the lower brainstem is responsible for this . Pure thalamic lesions, even those that cause a marked hemisensory loss, do not depress the corneal reflex.

Disturbances of Ocular Motility

Lesions restricted to the thalamus cause only subtle changes in ocular motility. Visual information from the superior colliculus, relayed by the pulvinar to the parietal lobe , contributes to the detection and localization of visual events in space and to the production of saccadic eye movements that allow the geniculostriate visual system to identify such events. Lesions in the pulvinar have been said to cause

(a) a decrease in the critical flicker frequency and neglect of visual objects in the periphery of the contralateral visual field,

(b) prolonged latency of visually evoked saccadic eye movements, and

(c) a paucity of spontaneous eye movements directed toward the contralateral hemifield.

Eye movement abnormalities that occur when a lesion involves the midbrain and thalamus; this often happens with paramedianthalamopeduncularinfarcts

[24]. So, impairment of ocular motor function results in abnormal pupils, ptosis, and restriction of vertical eye movements and of adduction. Selective upgaze, downgaze, or combined dysfunction may occur, as may blepharospasm, bilateral internuclearophthalmoplegia with ptosis , and pseudo-sixth nerve palsy.

Thalamic lesions may be associated with vertical gaze palsies not due to thalamic injury per se but due to extension of the lesions into the upper midbrain. A vertical one-and-a-half syndrome i.e.vertical palsy in one eye, upward palsy in the other eye may occur . Bilateral medial thalamic lesions may cause purely vertical saccadic apraxia, affecting volitional vertical but not random saccades. Acute thalamic esotropia, with impaired upward gaze, can occur with infarction of the contralateral posterior thalamus in the basilar-communicating artery territory. Tonic activation of the medial rectus in this case could result from damage to direct inhibitory projections from the thalamus or impairment of inputs to midbrain neurons involved with vergence control.

Large thalamic hemorrhages may impinge on the midbrain or impair its function by causing raised intracranial pressure. The eyes then become tonically deviated down and slightly adducted, as if peering at the tip of the nose. In some instances, the eyes may be tonically deviated to the side of the

hemiparesis, opposite a thalamic hemorrhagei.e.wrong-way eyes. This finding has also been reported in extrathalamicsupratentorial lesions.

DBS for the treatment of epilepsy, with electrodes placed at the mesodiencephalic junction, just inferior to the centromedian nucleus of the thalamus, may cause nystagmus with constant velocity slow phases, beating to the right when the left thalamus is stimulated and vice versa.

Depression of the reticular activating system or involvement of both thalami results in small pupils (1 mm in diameter) that react well to light i.e.diencephalic pupils. Anisocoria is occasionally present, with the smaller pupil ipsilateral to the thalamic lesion.

Disturbances of Complex Sensorimotor Functions

The thalamus modulates the association cortex involved in the processing of language and other higher cortical functions, unilateral thalamic lesions do not impair these functions as much; the pattern of impairment has some localizing value. The contralateral motor neglect caused by lesions in the ventral lateral nucleus of the thalamus and the contralateral visual inattention that results from lesions in the pulvinar.

Patients with right thalamic lesions may have constructional apraxia and display marked neglect of the left hemifield. This hemineglect may be

associated with anosognosia and asomatognosia, thereby mimicking a parietal lobe lesion[12], and is likely due to damage to the intralaminar and ventrolateral nuclei on the right. Right thalamic lesions can also cause impairment in the identification of emotional facial expressions with preserved discrimination of facial identity i.e.prosopoaffectiveagnosia. Alexia related to impaired visuospatial perception has been described after right thalamo-occipital infarction.

Dominant-hemisphere thalamic lesions may cause a transient language disturbance i.e.thalamic aphasia characterized by

(a) reduced spontaneous speech with paraphasic errors and perseveration,

(b) varying degrees of auditory comprehension impairment,

(c) preserved repetition and reading,

(d) defective spontaneous writing and writing to dictation but normal copying,

(e) word-production anomia but spared word selection and word symbolism, and

(f) distractibility.

This deficit, called as a mixed transcortical aphasia, tends to improve in a few weeks [21]. The language deficit may have more semantic components when

the lesion is posterior and more motor components when the lesion affects the anterior nucleus.

Some patients have hypophonia and dysarthria. Such language impairment resembles the syptom that results from left medial frontal lesions. Other dorsomedial thalamic lesions cause a bizarre language pattern, with some characteristics of dysfunction in the prefrontal cortex. It contains intrusions and other evidence of temporal gating impairment, such as giving biographical information while working on a calculation test. Apraxicagraphia has been described with adorsomedial thalamic lesion.

The language impairment noted with left thalamic lesions and the visual neglect noted with right thalamic lesions are associated with decreased activity of ipsilateral frontal or temporoparietal association cortex .Deafferentation of an otherwise intact cortex due to the thalamic lesion may explain these cortical syndromes with thalamic lesions.

A stuttering-like repetitive speech disorder may occur with infarction in the paramedian thalami and midbrain. The compulsive repetitions, constant rate, and monotonous tone seen with these infarctions are not associated with ordinary stuttering.

Disturbances of Executive Function

Paramedian thalamic infarction may cause a lack of initiative and frontal lobe utilization behavior , suggesting a thalamofrontal component to environmental interactions that requires inhibition, self-monitoring, and cognitive flexibility. Failure of goal-directed regulation of behavior has been observed with a lesion affecting the right anteroventral thalamic region. A left-sided discrete infarction of the medial thalamus may cause severe impairment of complex executive behaviors, due to dysfunction of thalamofrontal linkages that help modulate complex human behavior. Repetitive movements clonic perseveration may be a manifestation of executive function disorder with thalamic lesions.

Infarction of the dorsomedial nucleus, intralaminar nuclei, and medial part of the ventrolateral nuclei is often associated with marked hypoperfusion of the overlying frontal region cortex on position emission tomography (PET) or single photon emission computed tomography (SPECT) These nuclei provide an important pathway for the information from the vegetative centers to reach the frontal lobe.

Topographic Localization of Thalamic Lesions

Anterior Thalamic Region

Discrete lesions can be silent or cause language disturbances when they affect the dominant hemisphere. They can also cause inattention, which results more often when the right hemisphere is involved. Bilateral lesions may cause akinesia, amnesia, and attentional disturbances. If the Lesions extends to the subthalamicarea , it may cause athetosis, chorea, or postural abnormalities i.e.thalamic hand.

Medial Thalamic Region

Lesions in this location can go unnoticed when they are small and unilateral. Large or bilateral lesions cause impairment of recent memory, apathy or agitation, attention derangements, and somnolence or coma. Lesions that extend to the midbrain-diencephalic junction can cause contralateral tremor and vertical gaze palsy, affecting particularly downward gaze.

Ventrolateral Thalamic Region

Sensory loss, paroxysmal pains, and hemiataxia in the contralateral side of the body are the most common sequelae of lesions in the posterior portion of this region. More anterior lesions can cause postural abnormalities, such as

disequilibrium and restriction of axial supportive movements or delayed tremor. Hemineglect and language disturbances can appear transiently.

Posterior Region

Basal lesions in the region may cause hemianesthesia, thalamic pain, and also visual field defects. Dorsal lesions give rise to attentional disorders of the ipsilateral hemisphere, resulting in transient aphasia if the dominant hemisphere is involved. Few patients may have myoclonic dystonia.

MATERIALS AND METHODS

This study was conducted from December 2012to January 2014. Patients were chosen from neurology and medical departments in Rajiv Gandhi Government General hospital ,chennai. They were enrolled in this study after getting a written consent.

INCLUSION CRITERIA

1) Patients who presented with symptoms and signs of thalamic stroke.

2) Patients who presented with both ischemic and haemorrhagic thalamic stroke.

3) Patients who underwent both CT scan brain and MRI brain

EXCLUSION CRITERIA :

1)Patients who presented with symptoms and signs of stroke in other regions.

2)Patients who had previous stroke.

3)Patients who were not done computed tomography (CT) brain& MRI brain

4)Patients who were having contraindications to Magnetic Resonance Imaging.

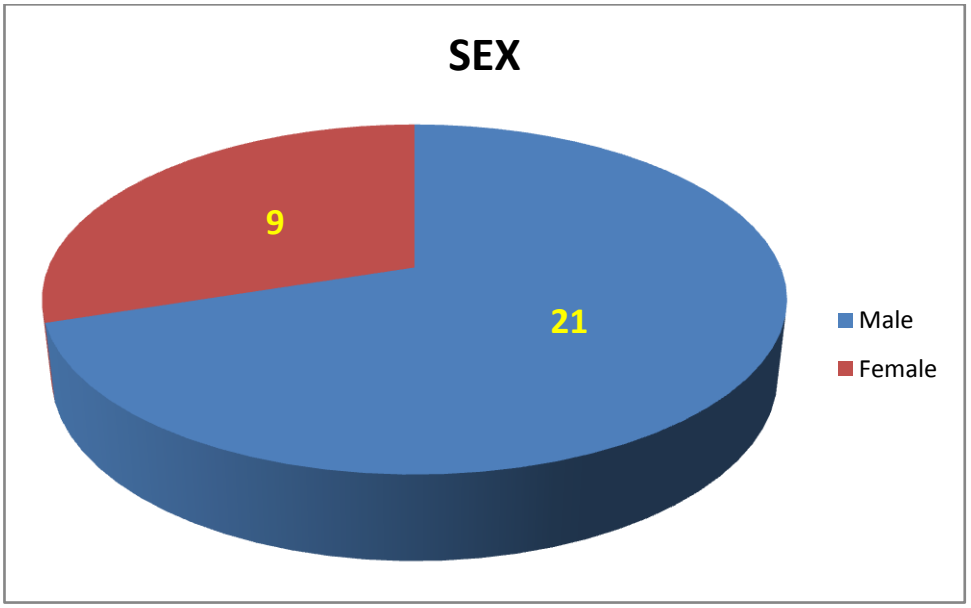
Patients who presented with stroke symptoms and signs were subjected to plain CT Brain and Magnetic Resonance Imaging (MRI) and MRA.

OBSERVATIONS AND RESULTS

SEX DISTRIBUTION

In this study, out of 30 patients 21 (70%) were males and 9 (30%) were females (Figure-1).

SEX	Number of Patients	Percent
Male	21	70
Female	9	30
Total	30	100

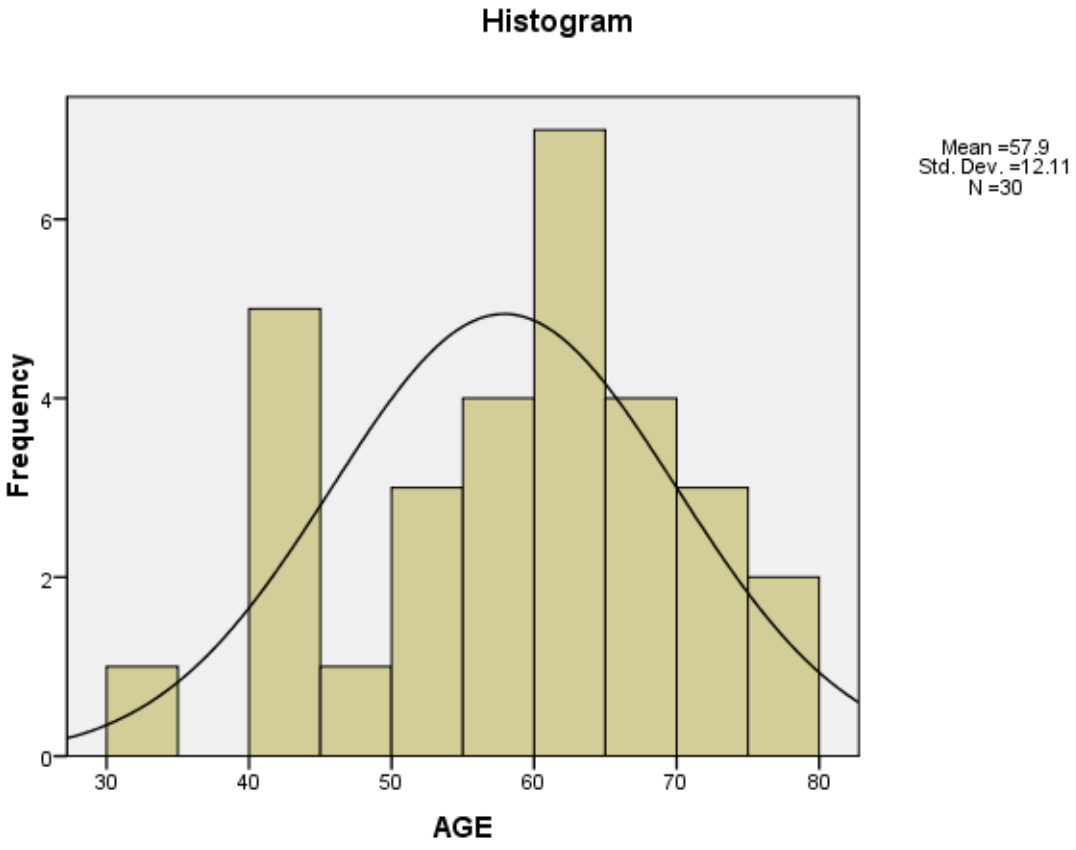


AGE DISTRIBUTION

In this study, out of 30 patients, 1 (3.3%) patients were in the age group of less than 40 years of their age. 6 (20%) patients were between 40 to 50 years of age. 7 (23.3%) patients were between 50-60 years of age.11(36.7%)were between 60-70 yrs of age. 5(16.6%) were between

70 -80 yrs of age (Table-2 and Figure-2).

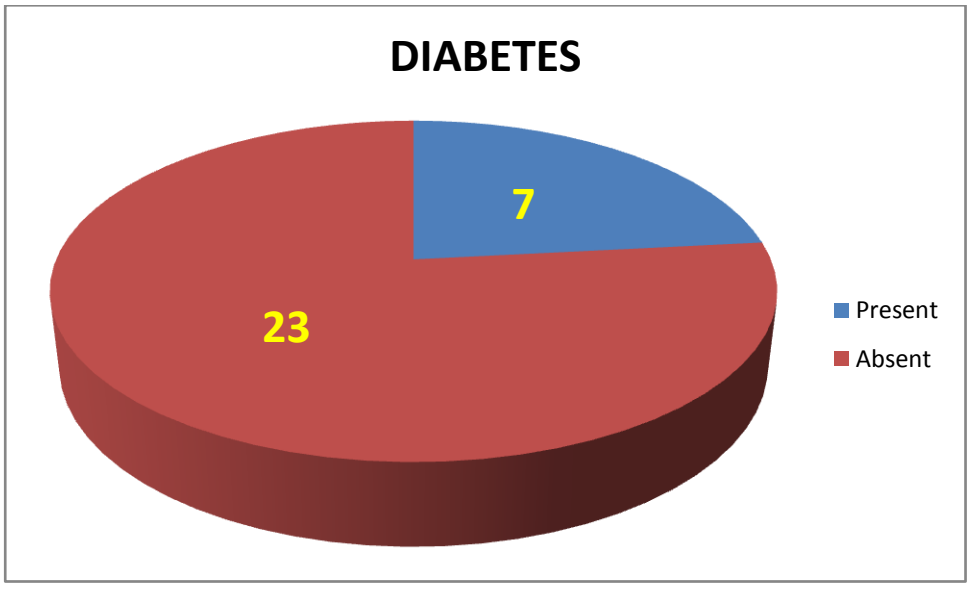
Age Distribution				
	N	Minimum	Maximum	Mean
AGE	30	30	78	57.90



DIABETES MELLITUS

In 30 stroke patients, 7 (23.3%) had diabetes mellitus. Out of 7patients 5 were on regular treatment and rest of the 2 patients were not on regular treatment. Among them, 6 patients were males and remaining 1of them is female (Figure-

DIABETES	Number of Patients
Present	7
Absent	23
Total	30

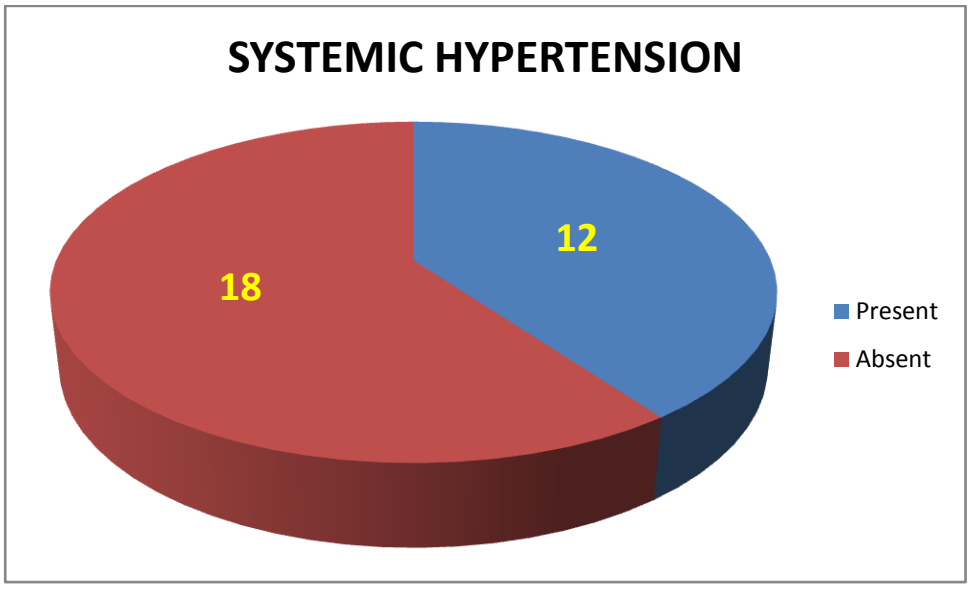


SYSTEMIC HYPERTENSION

Out of 30 patients, 12 (40%) were already known hypertensive patients. All of them were on regular treatment. Among them 2were female patients and the remaining 10 were male patients (Table3- and Figure-3).

Thalamic haemorrhage was present in 5 (50%) of hypertensive patients.

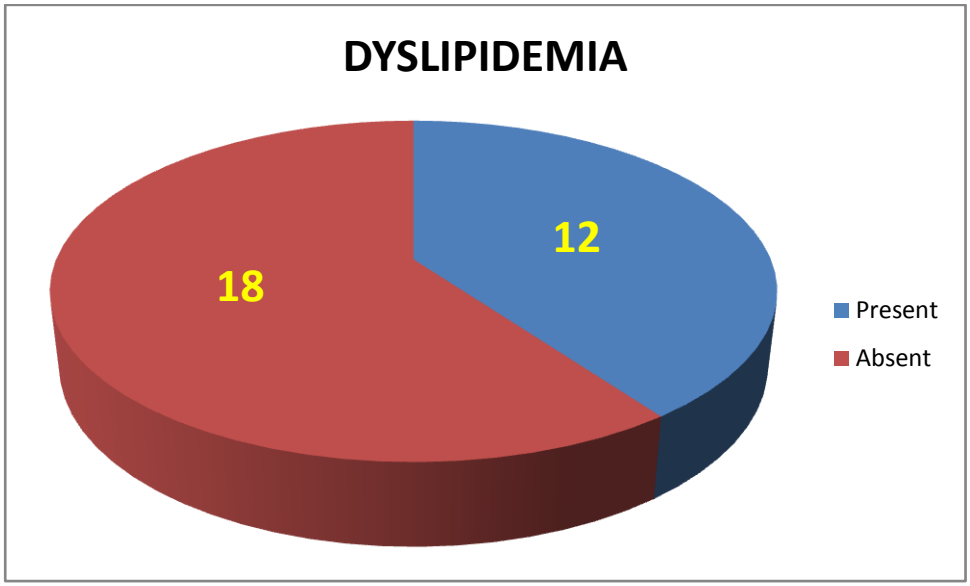
SYSTEMIC HYPERTENSION	Number of Patients	Percent
Present	12	40
Absent	18	60
Total	30	100



DYSLIPIDEMIAS

In this study out of 30 patients, 12 (40%) patients had dyslipidemias. Out of 12 patients, 7 being female patients and rest of the 5 were male patients (Table- and Figure-)

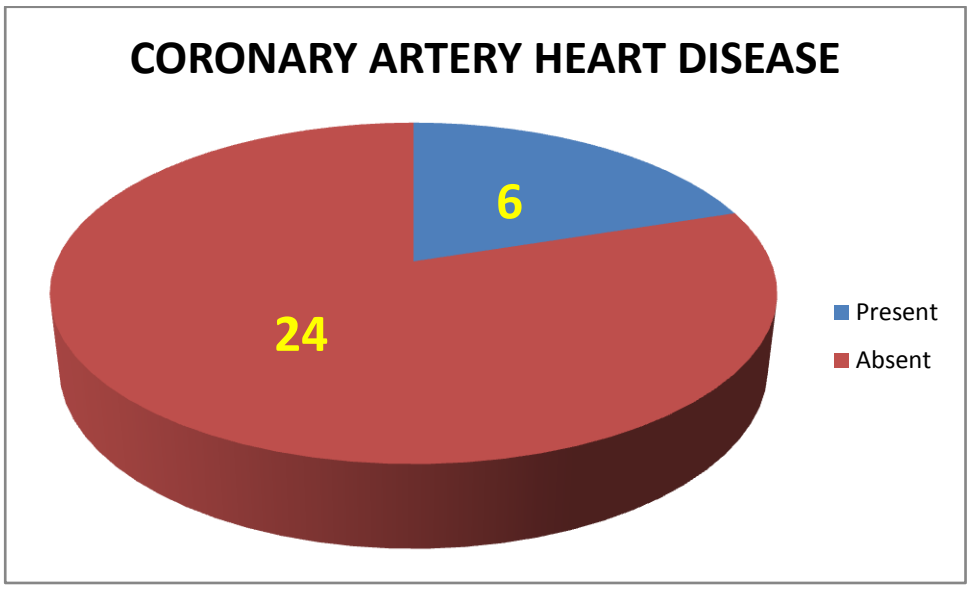
DYSLIPIDEMIA	Number of Patients	Percent
Present	12	40
Absent	18	60
Total	30	100



CORONARY ARTERY HEART DISEASE

6 (20%) patients were having coronary artery heart disease. 5 were on treatment already. One of them was diagnosed after admission (table).

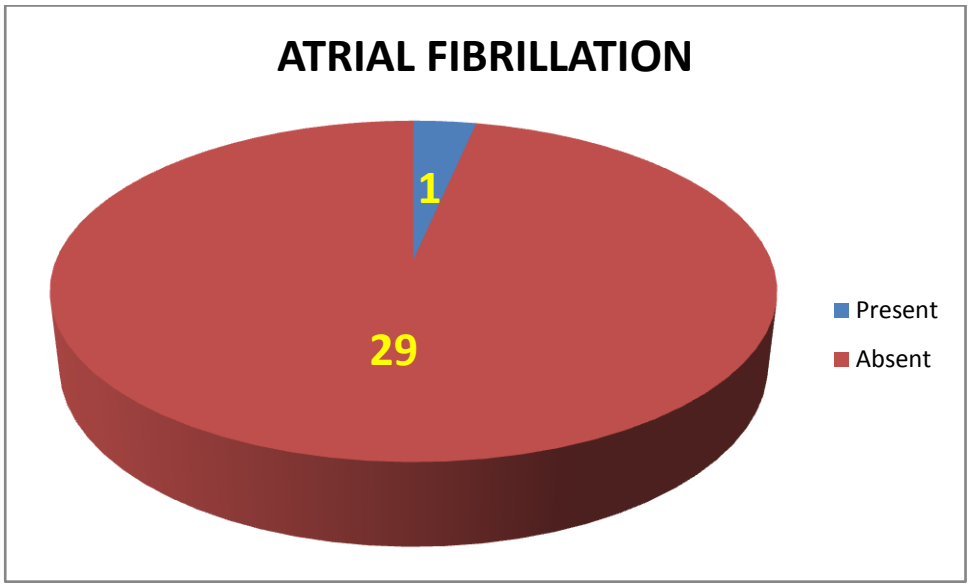
CORONARY ARTERY HEART DISEASE	Number of Patients	Percent
Present	6	20.0
Absent	24	80.0
Total	30	100.0



ATRIAL FIBRILLATION

In this study out of 30 patients, 1 male patient with thalamic infarct had atrial fibrillation.

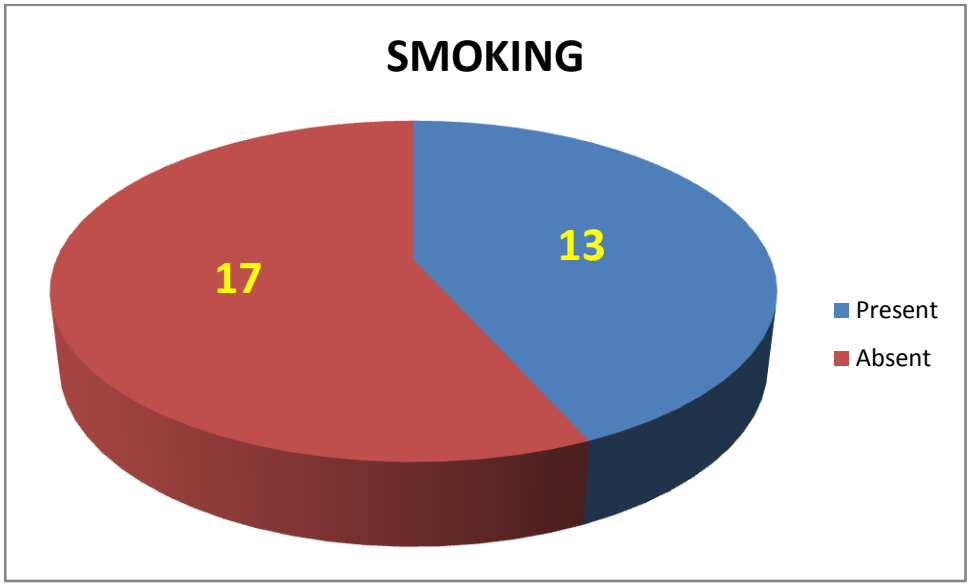
ATRIAL FIBRILLATION	Number of Patients	Percent
Present	1	3.3
Absent	29	96.7
Total	30	100



SMOKING

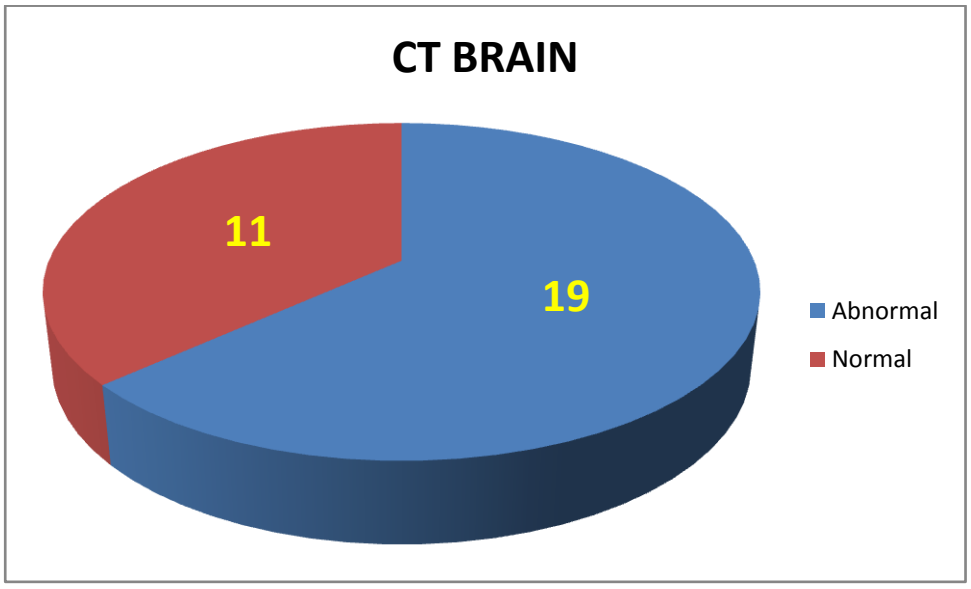
In this study of 30 pts, 13 (43.3%) patients , who all are male has positive history of chronic smoking. Out of this , 10 patients had ischemic stroke.

SMOKING	Number of Patients	Percent
Present	13	43.3
Absent	17	56.7
Total	30	100



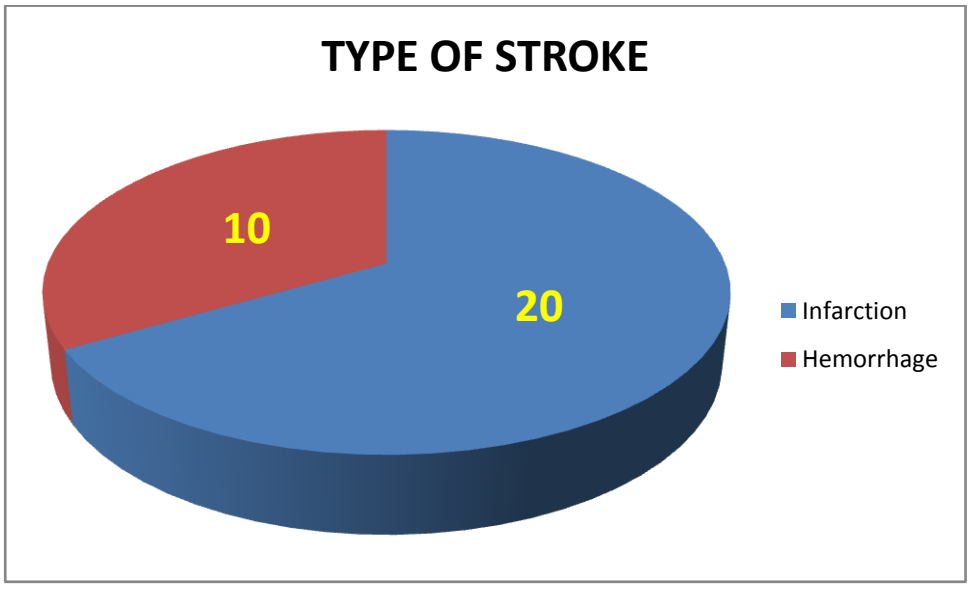
CT BRAIN

CT BRAIN	Number of Patients	Percent
Abnormal	19	63.3
Normal	11	36.7
Total	30	100



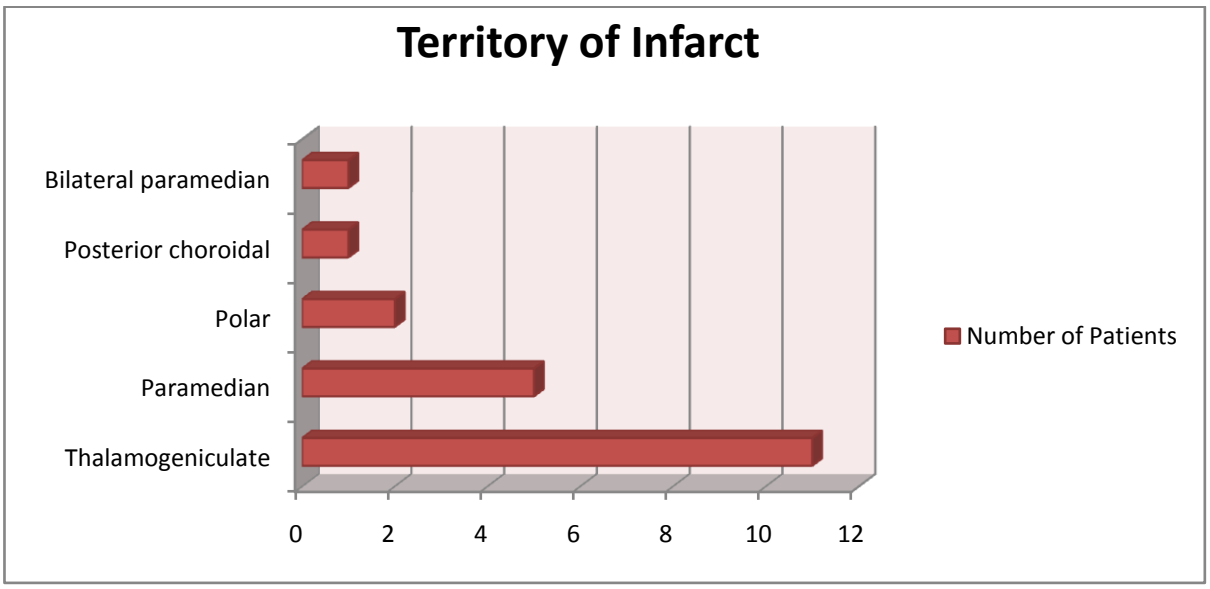
TYPE OF STROKE

TYPE OF STROKE	Number of Patients	Percent
Infarction	20	66.7
Hemorrhage	10	33.3
Total	30	100



TERRITORY OF INFARCT

TERRITORY OF INFARCT	Number of Patients	Percent
Thalamogeniculate	11	55.5
Paramedian	5	25.5
Polar	2	10
Posterior choroidal	1	5
Bilateral paramedian	1	5



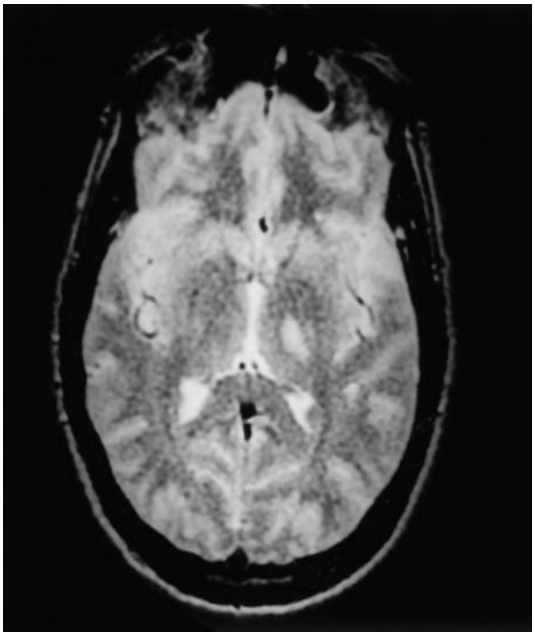


Figure 1. T2-weighted MRI scan showing left thalamogeniculate (inferolateral) infarction.

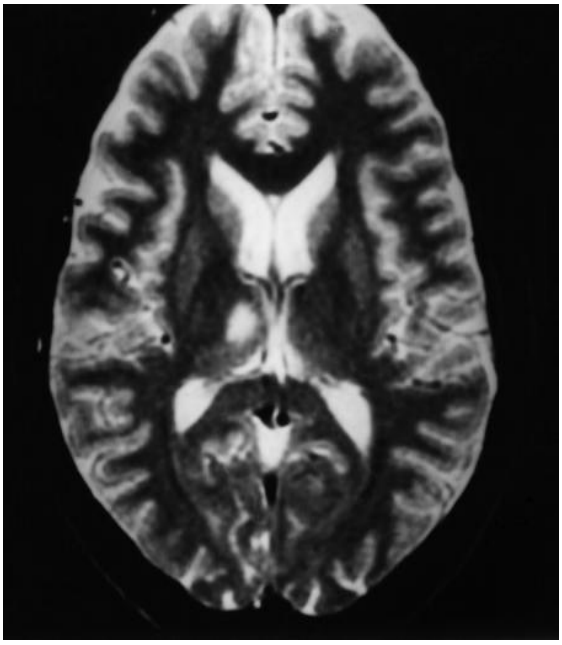


Figure 2. T2-weighted MRI scan showing unilateral right paramedian infarction.

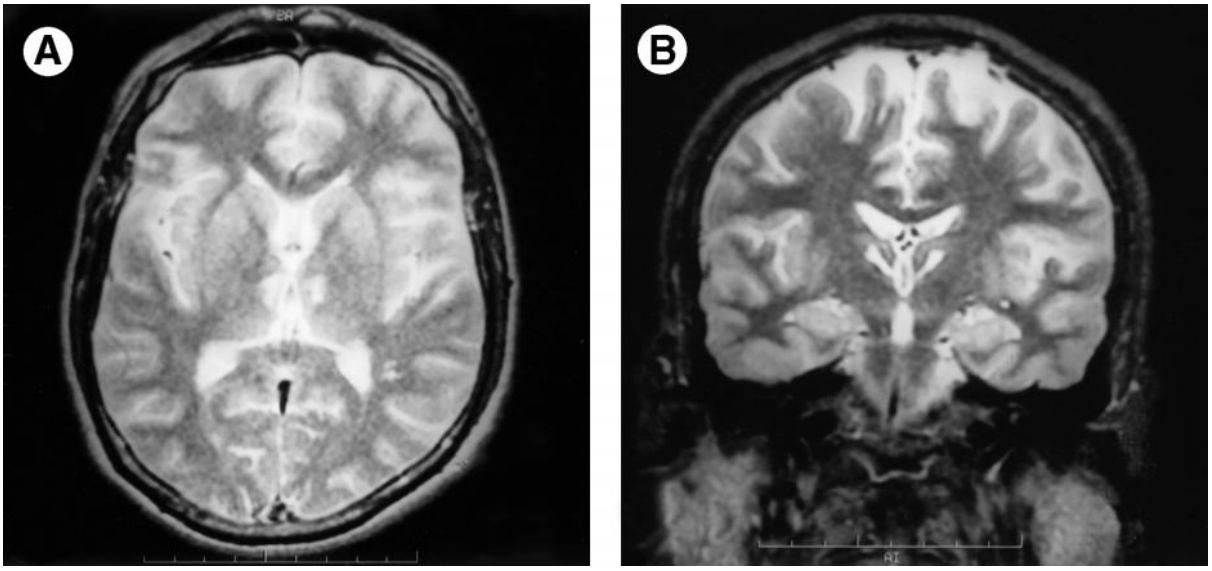


Figure 3. T2-weighted MRI scans showing bilateral paramedian infarction. (A) Axial view. (B) Coronal view.

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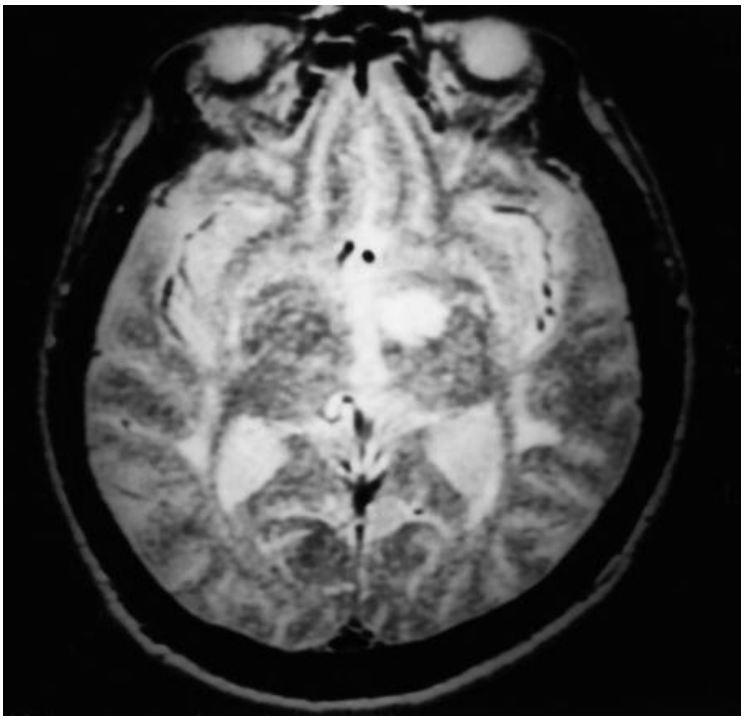


Figure 4. T2-weighted MRI scan showing unilateral left polar (tuberothalamic)

infarction.

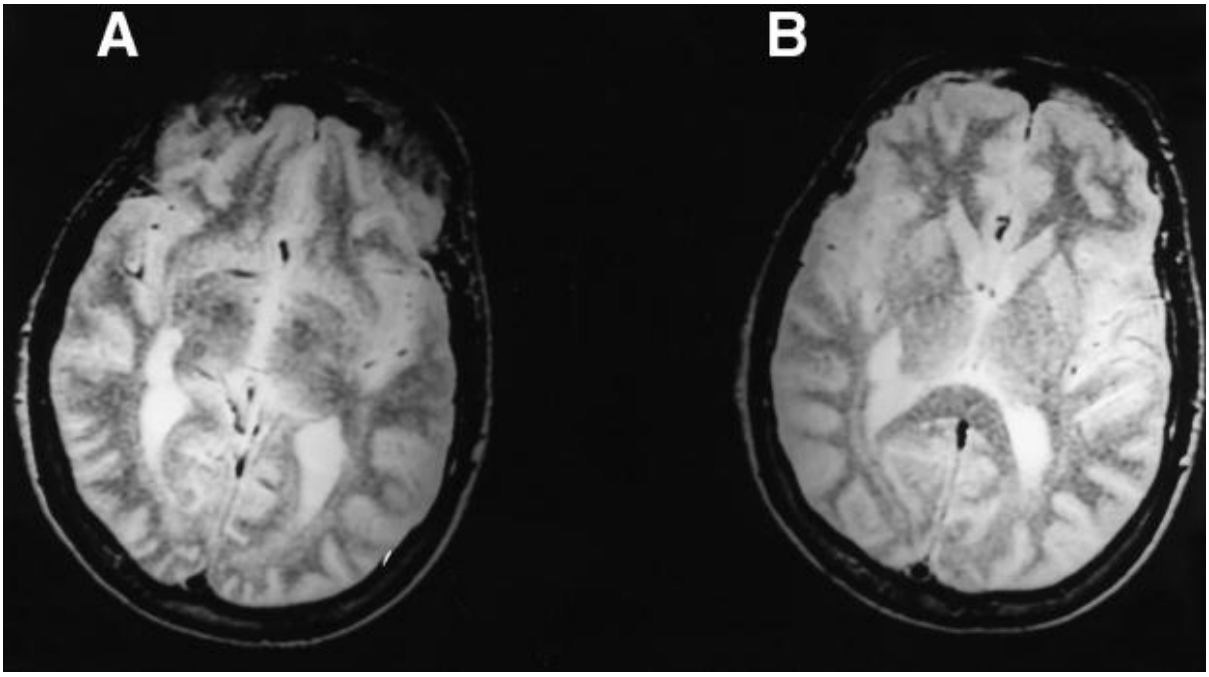
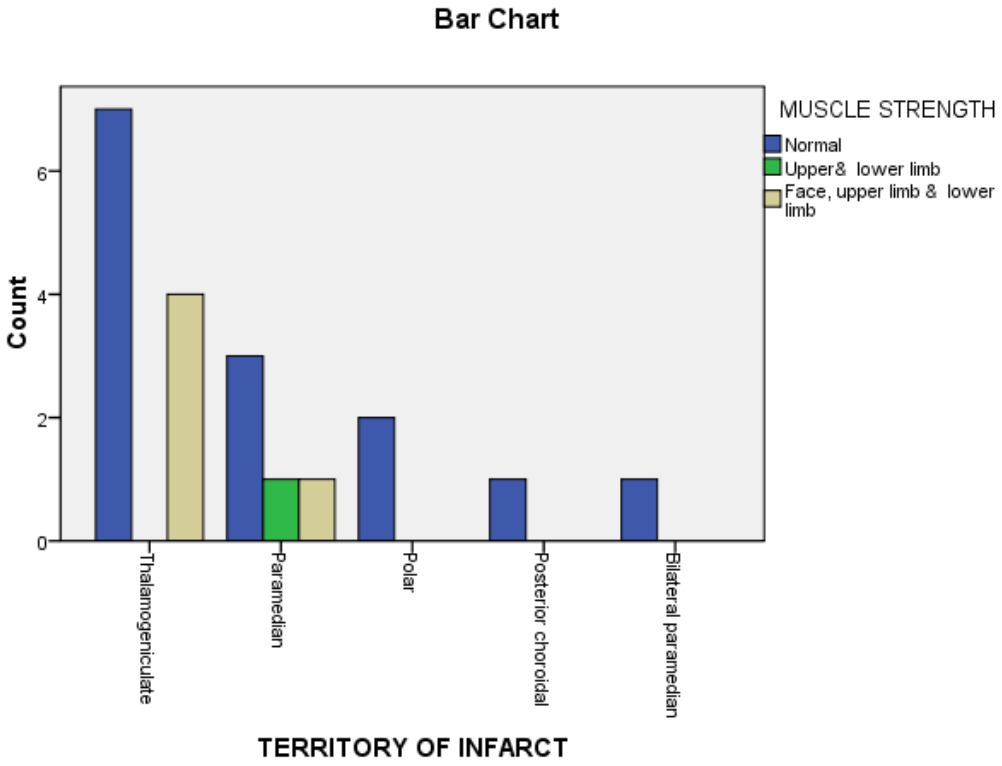


Figure 5. T2-weighted MRI scans showing unilateral posterior choroidal infarction.

CLINICORADIOLOGICAL CORRELATION - INFARCTION

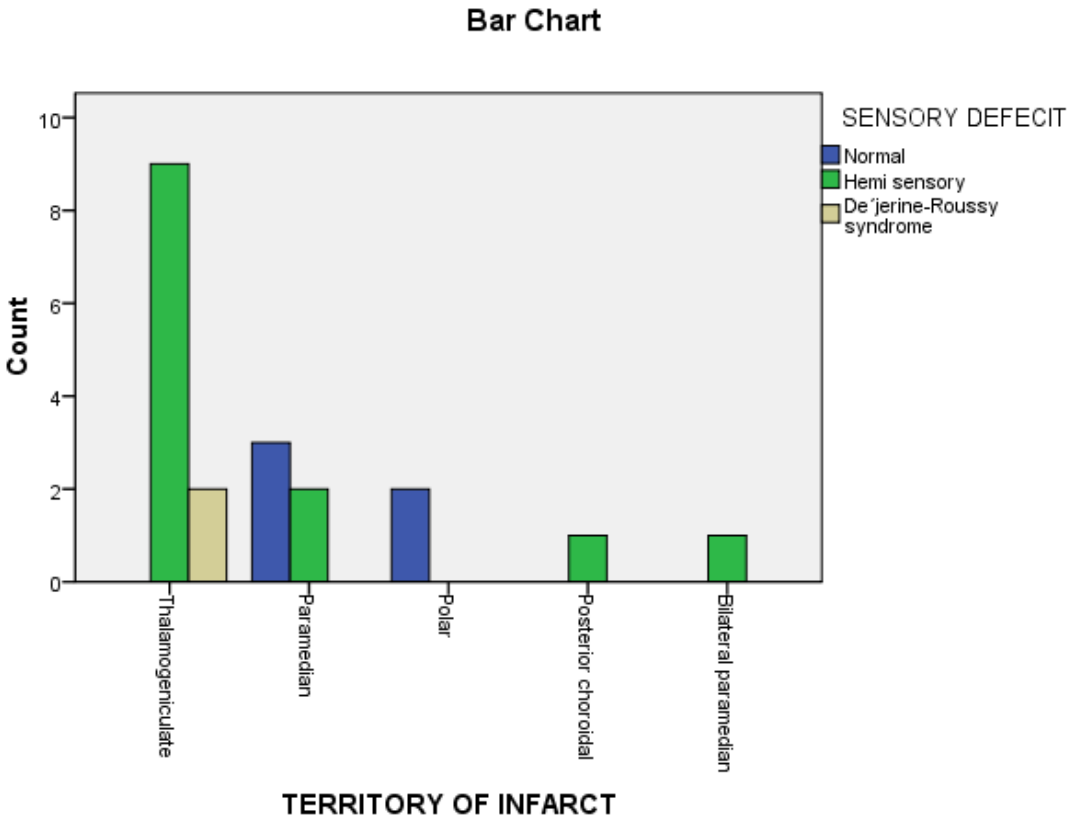
Pattern of Weakness

TERRITORY OF INFARCT	Pattern of Weakness		
	Normal	Upper& lower limb	Face, upper limb & lower limb
Bilateral paramedian	1	0	0
	5.0%	.0%	.0%
Posterior choroidal	1	0	0
	5.0%	.0%	.0%
Polar	2	0	0
	10.0%	.0%	.0%
Paramedian	3	1	1
	15.0%	5.0%	5.0%
Thalamogeniculate	7	0	4
	35.0%	.0%	20.0%
Count	14	1	5
% of Total	70.0%	5.0%	25.0%



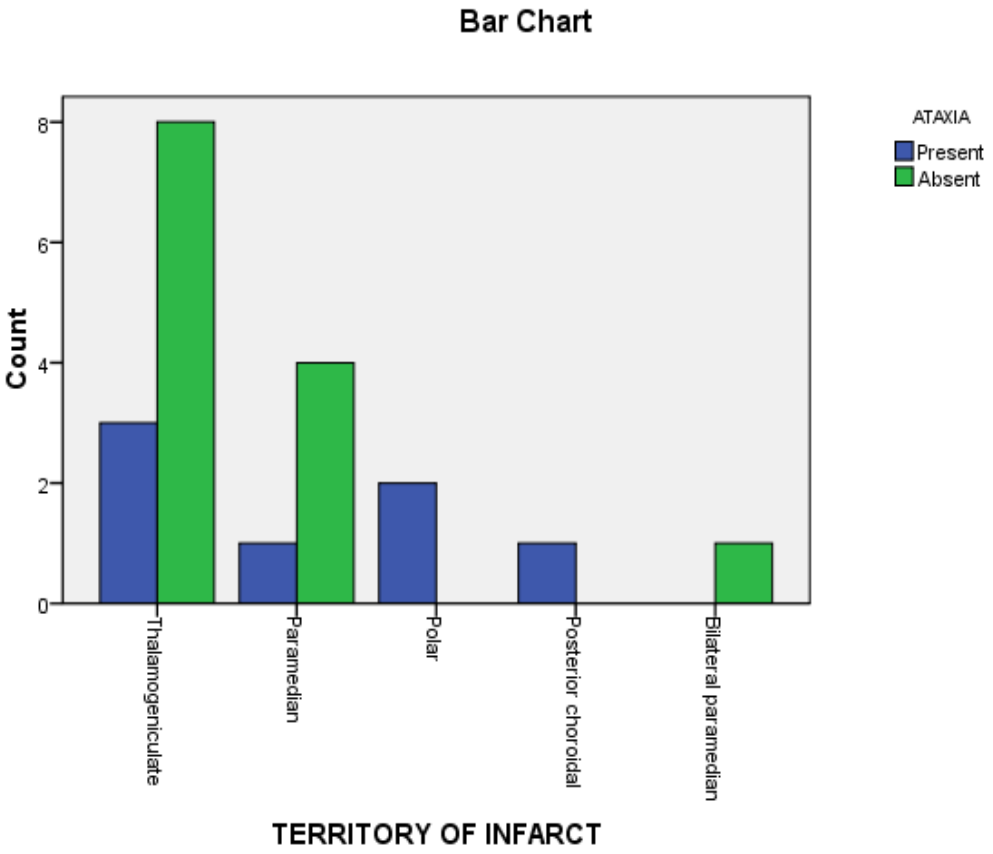
Pattern of Sensory Deficit

TERRITORY OF INFARCT	Pattern of Sensory Deficit		
	Normal	Hemi sensory	De´jerine-Roussy syndrome
Bilateral paramedian	0	1	0
	.0%	5.0%	.0%
Posterior choroidal	0	1	0
	.0%	5.0%	.0%
Polar	2	0	0
	10.0%	.0%	.0%
Paramedian	3	2	0
	15.0%	10.0%	.0%
Thalamogeniculate	0	9	2
	.0%	45.0%	10.0%
Count	13	2	20
% of Total	65.0%	10.0%	100.0%



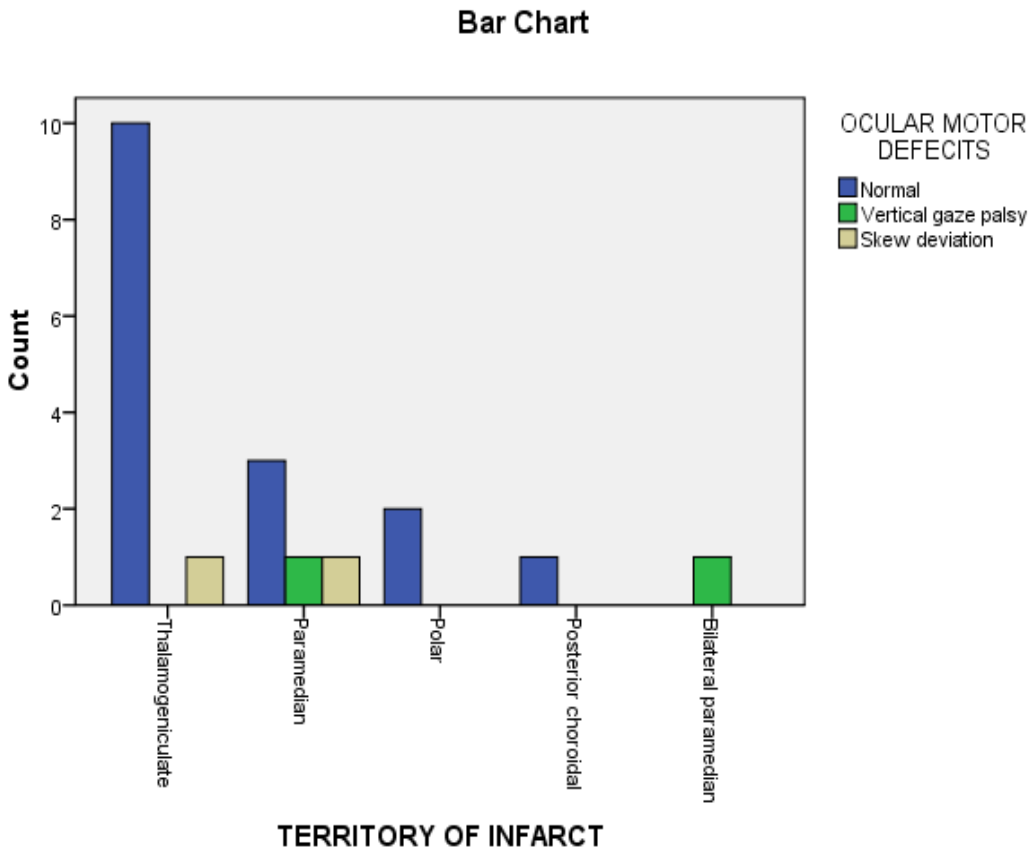
Ataxia

TERRITORY OF INFARCT	ATAXIA	
	Present	Absent
Bilateral paramedian	0	1
Posterior choroidal	1	0
Polar	2	0
Paramedian	1	4
Thalamogeniculate	3	8
Total	7	13



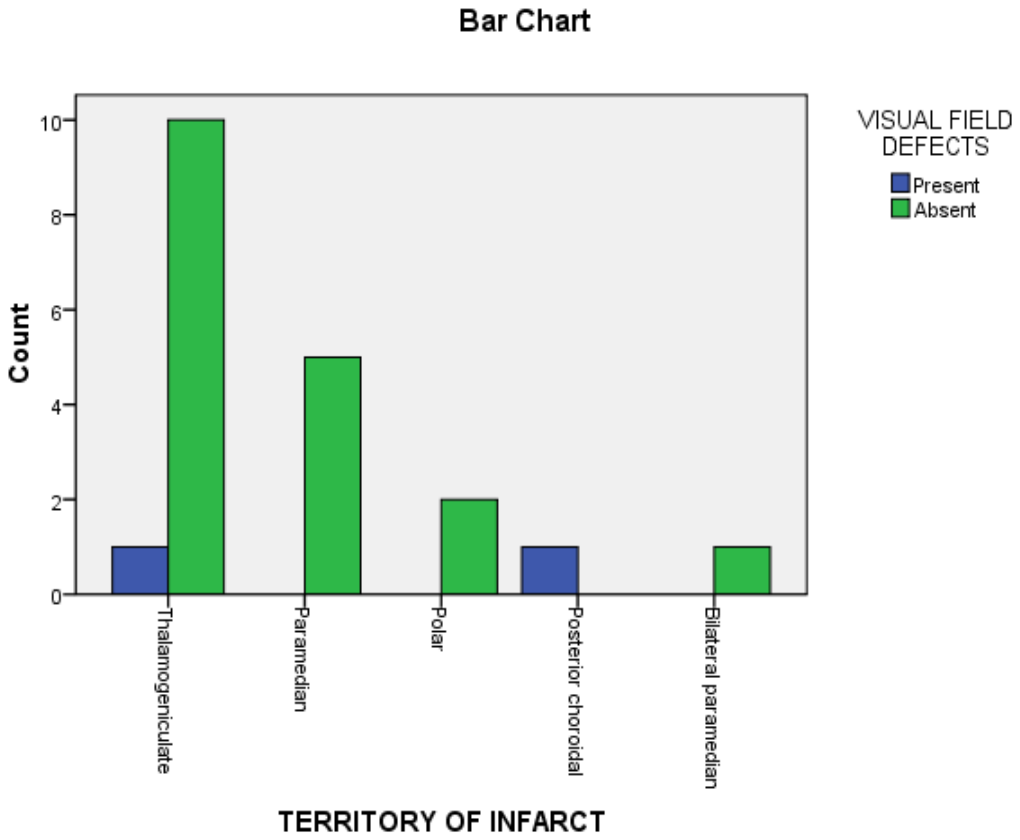
Ocular Motor Defecits

TERRITORY OF INFARCT	OCULAR MOTOR DEFECITS		
	Normal	Vertical gaze palsy	Skew deviation
Bilateral paramedian	0	1	0
Posterior choroidal	1	0	0
Polar	2	0	0
Paramedian	3	1	1
Thalamogeniculate	10	0	1
Total	16	2	2



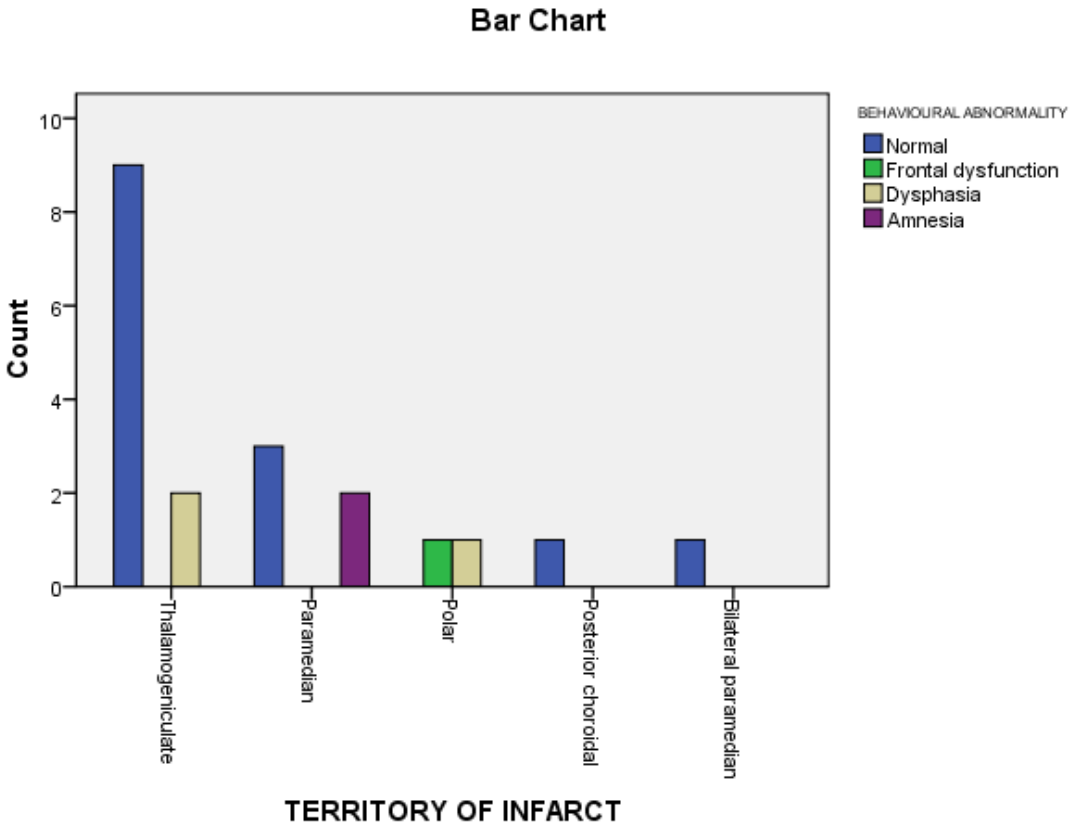
Visual Field Defects

TERRITORY OF INFARCT	VISUAL FIELD DEFECTS	
	Present	Absent
Bilateral paramedian	0	1
Posterior choroidal	1	0
Polar	0	2
Paramedian	0	5
Thalamogeniculate	1	10
Total	2	18



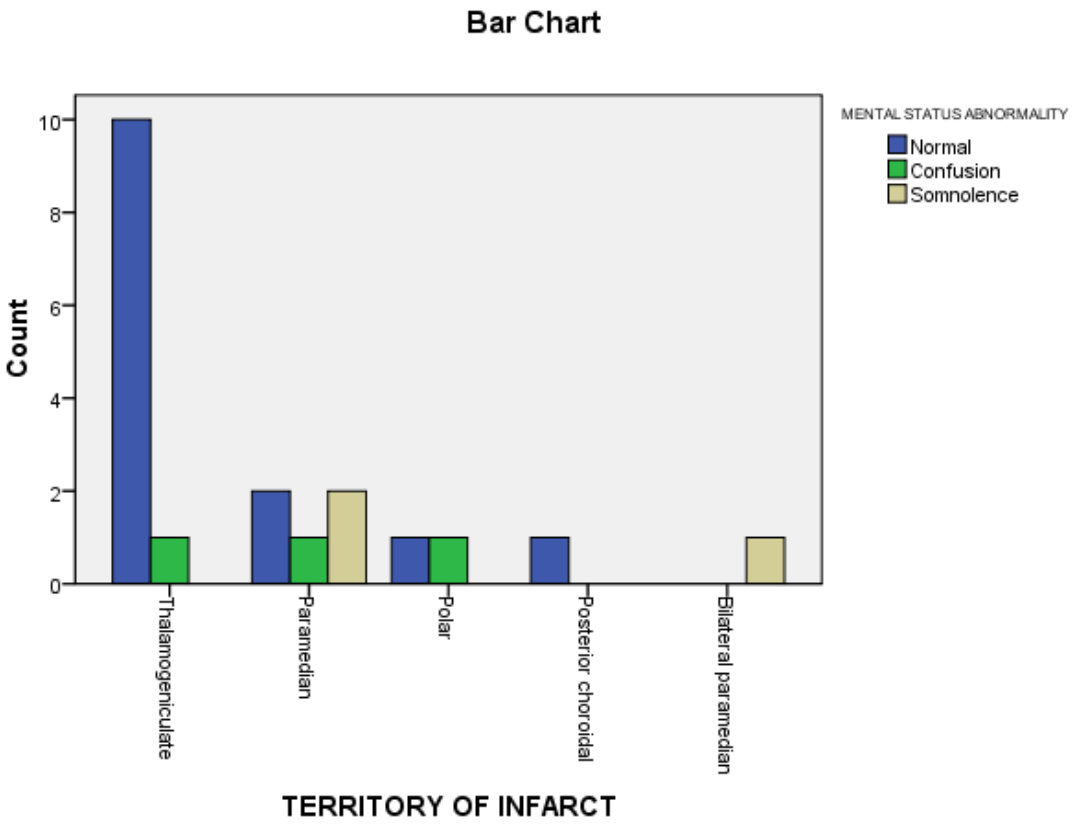
Behavioural Abnormality

TERRITORY OF INFARCT	BEHAVIOURAL ABNORMALITY			
	Normal	Frontal dysfunction	Dysphasia	Amnesia
Bilateral paramedian	1	0	0	0
Posterior choroidal	1	0	0	0
Polar	0	1	1	0
Paramedian	3	0	0	2
Thalamogeniculate	9	0	2	0
Total	14	1	3	2



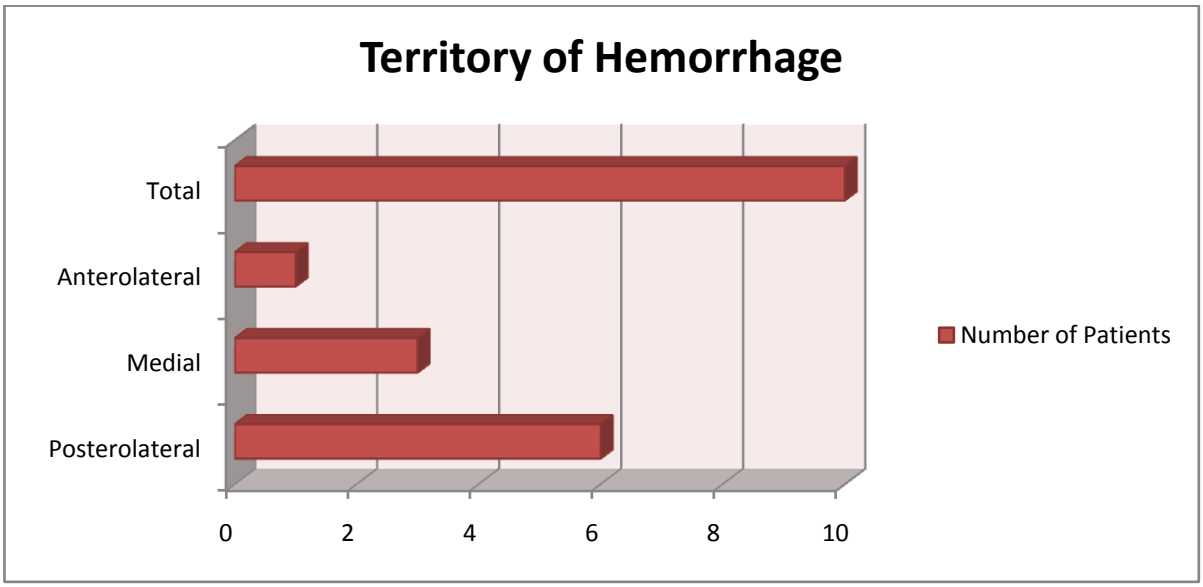
Mental Status Abnormality

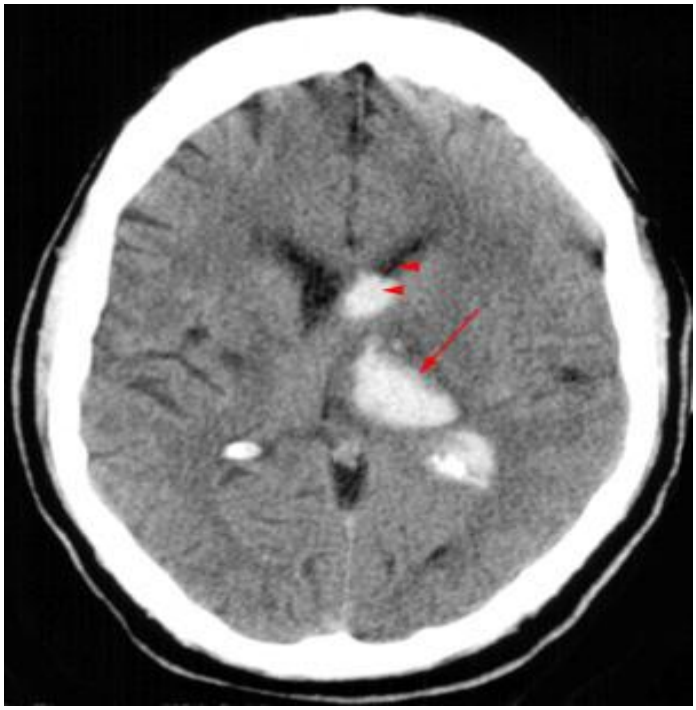
TERRITORY OF INFARCT	MENTAL STATUS ABNORMALITY		
	Normal	Confusion	Somnolence
Bilateral paramedian	0	0	1
Posterior choroidal	1	0	0
Polar	1	1	0
Paramedian	2	1	2
Thalamogeniculate	10	1	0
Total	14	3	3



TERRITORY OF HEMORRHAGE

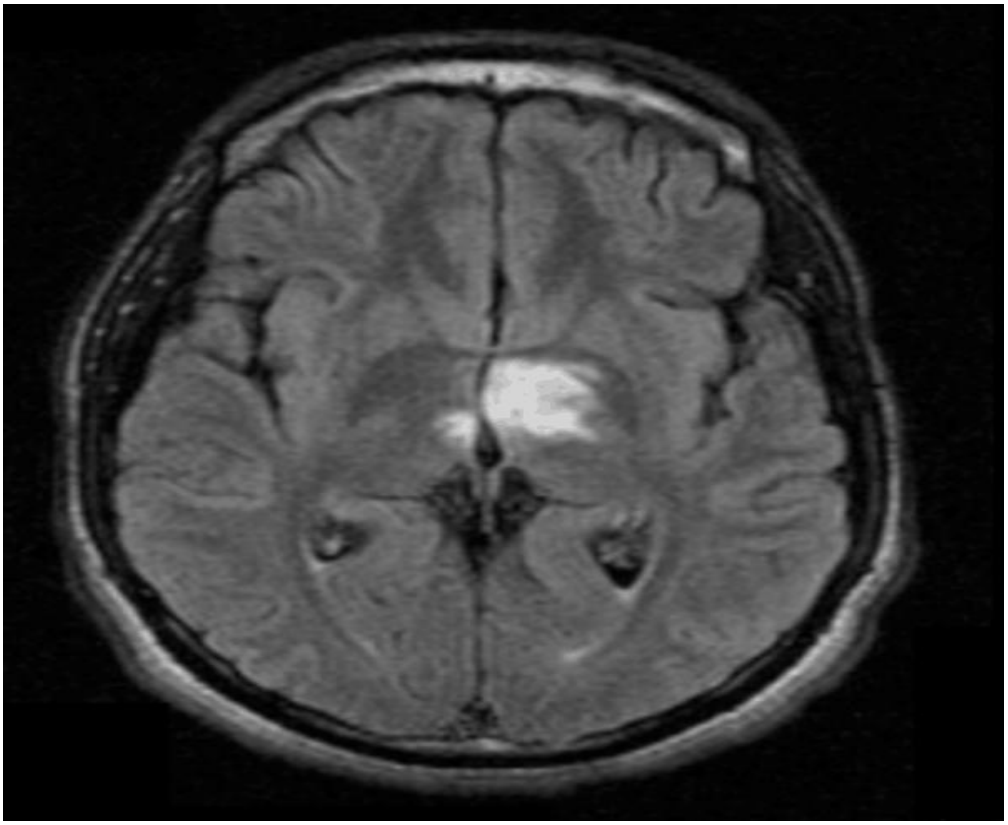
TERRITORY OF HEMORRHAGE	Number of Patients	Percent
Posterolateral	6	60
Medial	3	30
Anterolateral	1	10
Dorsal	0	0
Total	10	100





CT SCAN BARIN – LEFT THALAMIC HAEMORRHAGE & VENTRICULAR EXTENSION

CT BRAIN-RIGHT THALAMIC HAEMORRHAGE

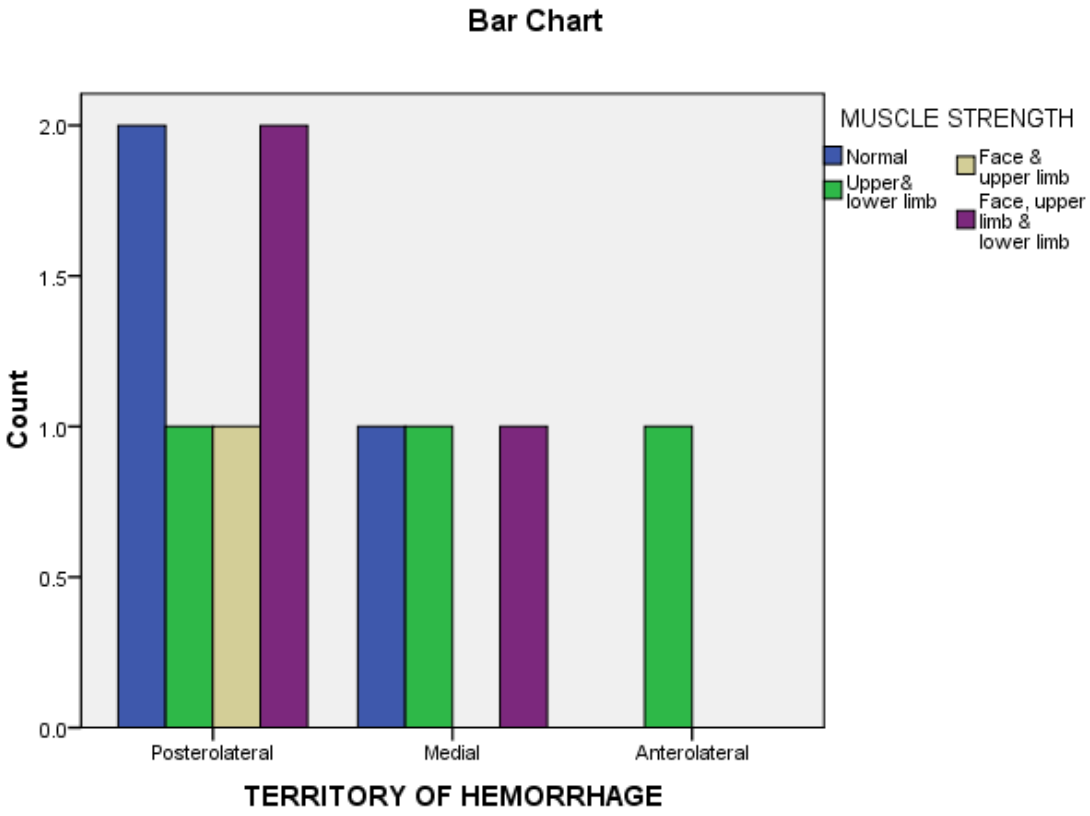


MRI BRAIN – LEFT ANTERIOR THALAMIC HAEMORRHAGE

CLINICORADIOLOGICAL CORRELATION – HEMORRHAGE

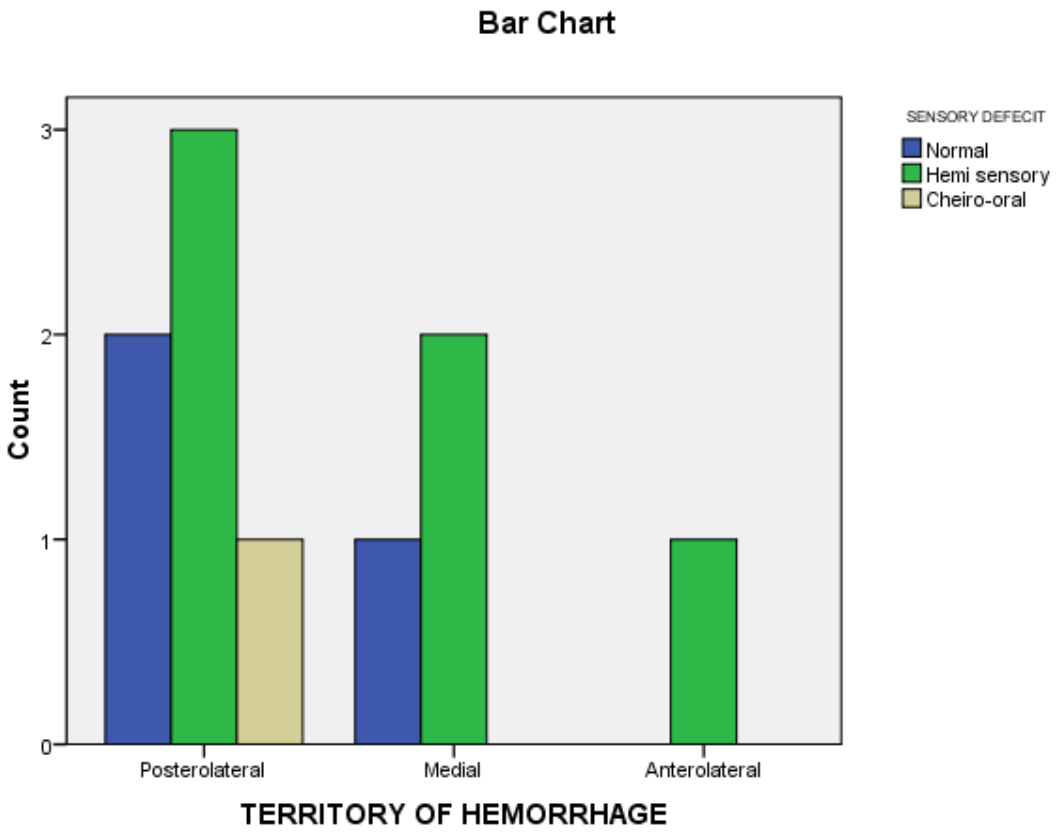
Pattern of Weakness

TERRITORY OF HEMORRHAGE	Pattern of Weakness			
	Normal	Upper& lower limb	Face & upper limb	Face, upper limb & lower limb
Anterolateral	0	1	0	0
Medial	1	1	0	1
Posterolateral	2	1	1	2
Dorsal	0	0	0	0
Total	3	3	1	3



Pattern of Sensory Deficit

TERRITORY OF HEMORRHAGE	SENSORY DEFECIT		
	Normal	Hemi sensory	Cheiro-oral
Anterolateral	0	1	0
Medial	1	2	0
Posterolateral	2	3	1
Dorsal	0	0	0
Total	3	6	1

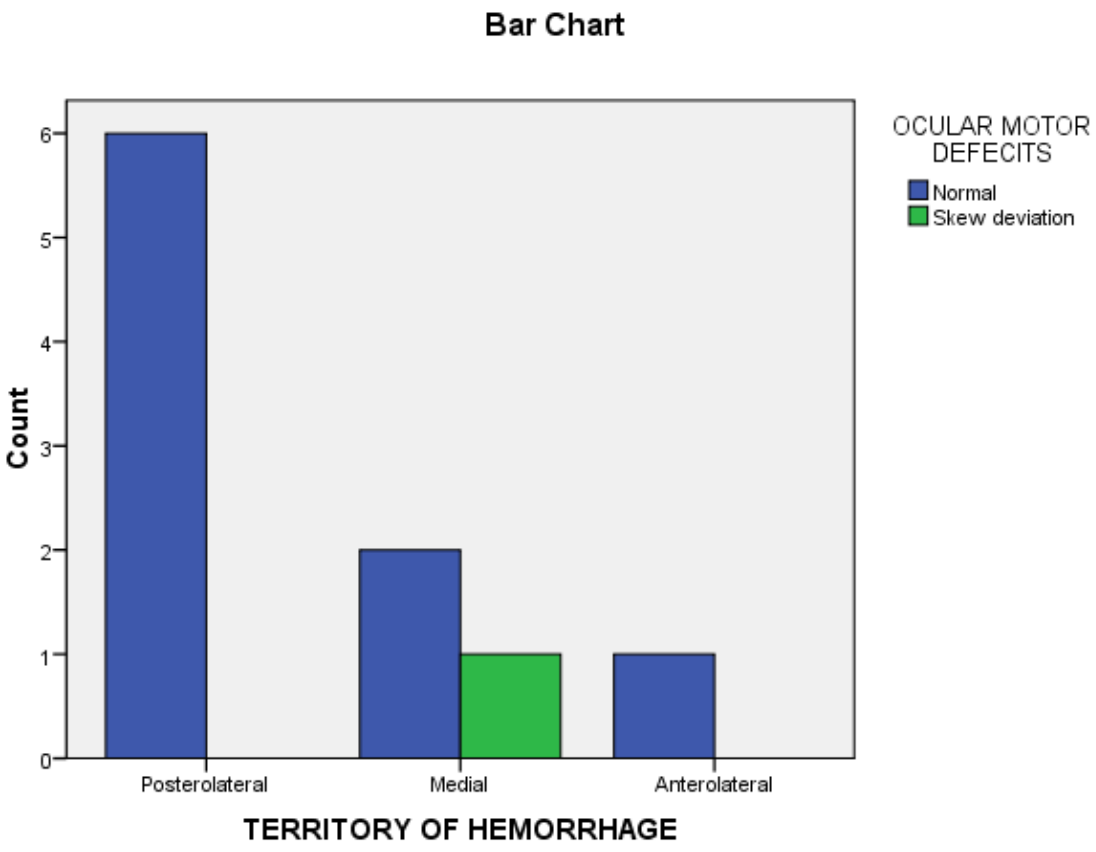


Ataxia

TERRITORY OF HEMORRHAGE	ATAXIA	
	Present	Absent
Anterolateral	1	0
Medial	1	2
Posterolateral	0	6
Dorsal	0	0
Total	2	8

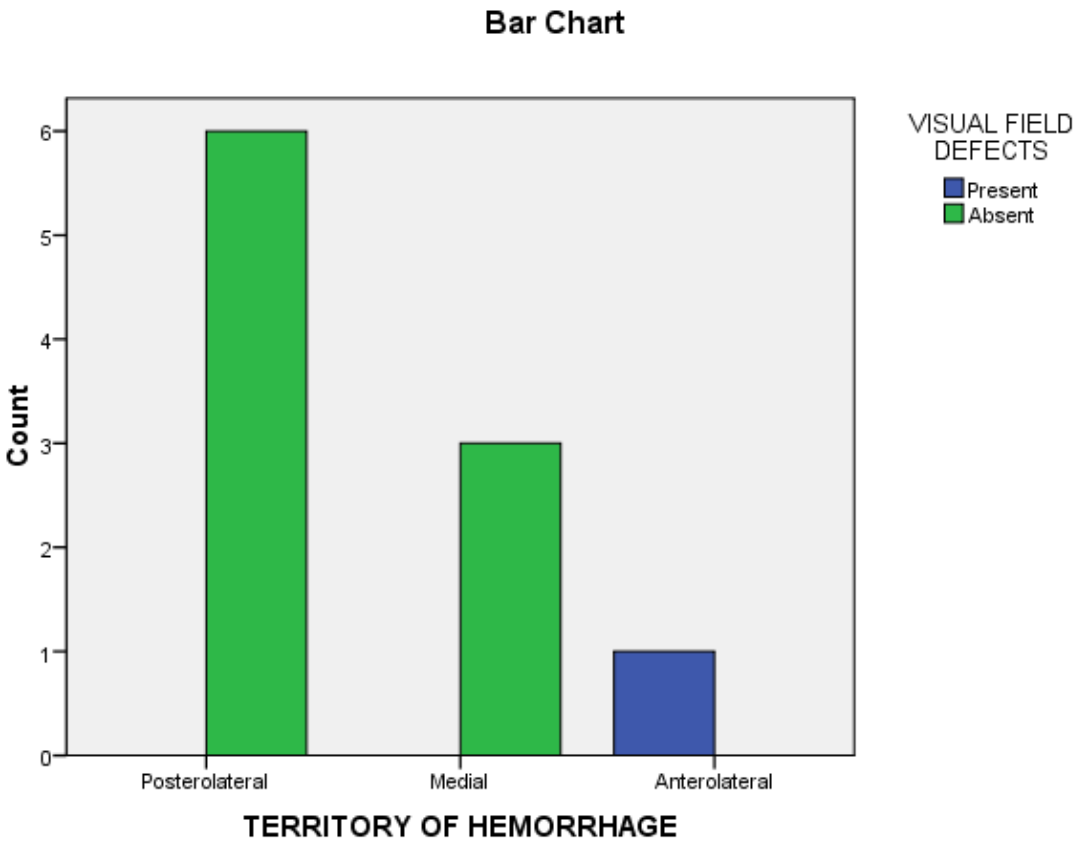
Ocular Motor Defecits

TERRITORY OF HEMORRHAGE	OCULAR MOTOR DEFECITS	
	Normal	Skew deviation
Anterolateral	1	0
Medial	2	1
Posterolateral	6	0
Dorsal	0	0
Total	9	1



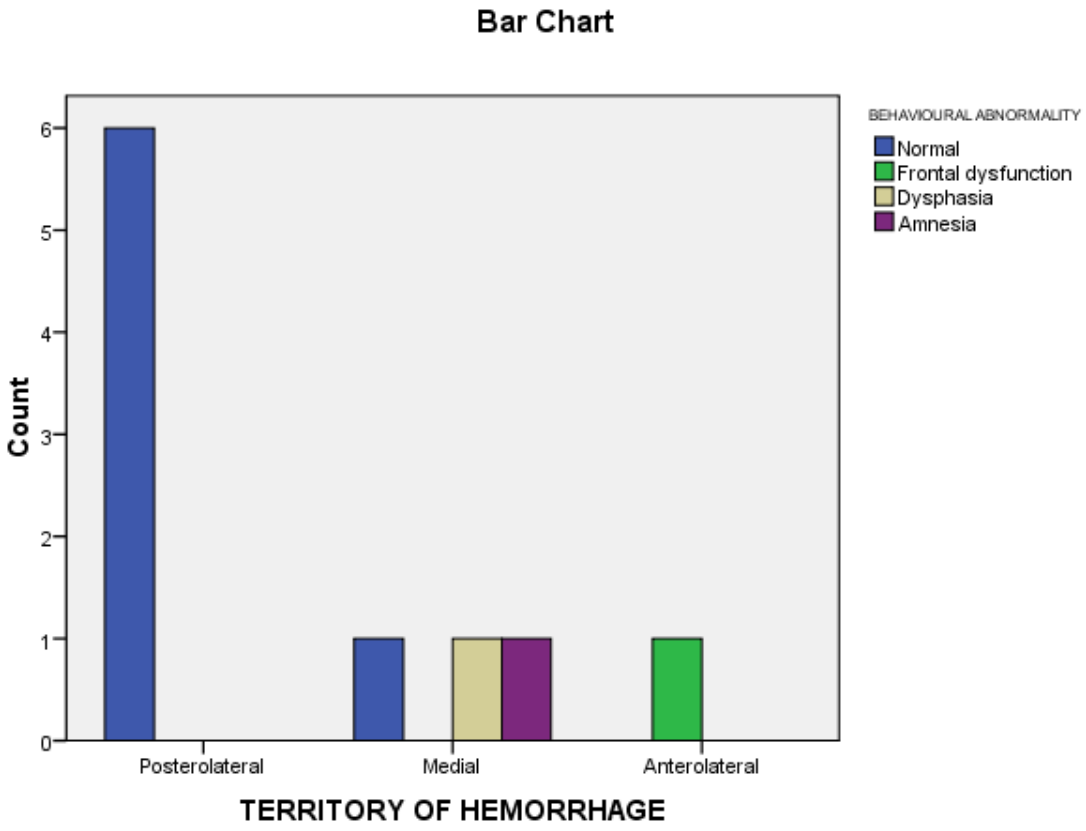
Visual Field Defects

TERRITORY OF HEMORRHAGE	VISUAL FIELD DEFECTS	
	Present	Absent
Anterolateral	1	0
Medial	0	3
Posterolateral	0	6
Dorsal	0	0
Total	1	9



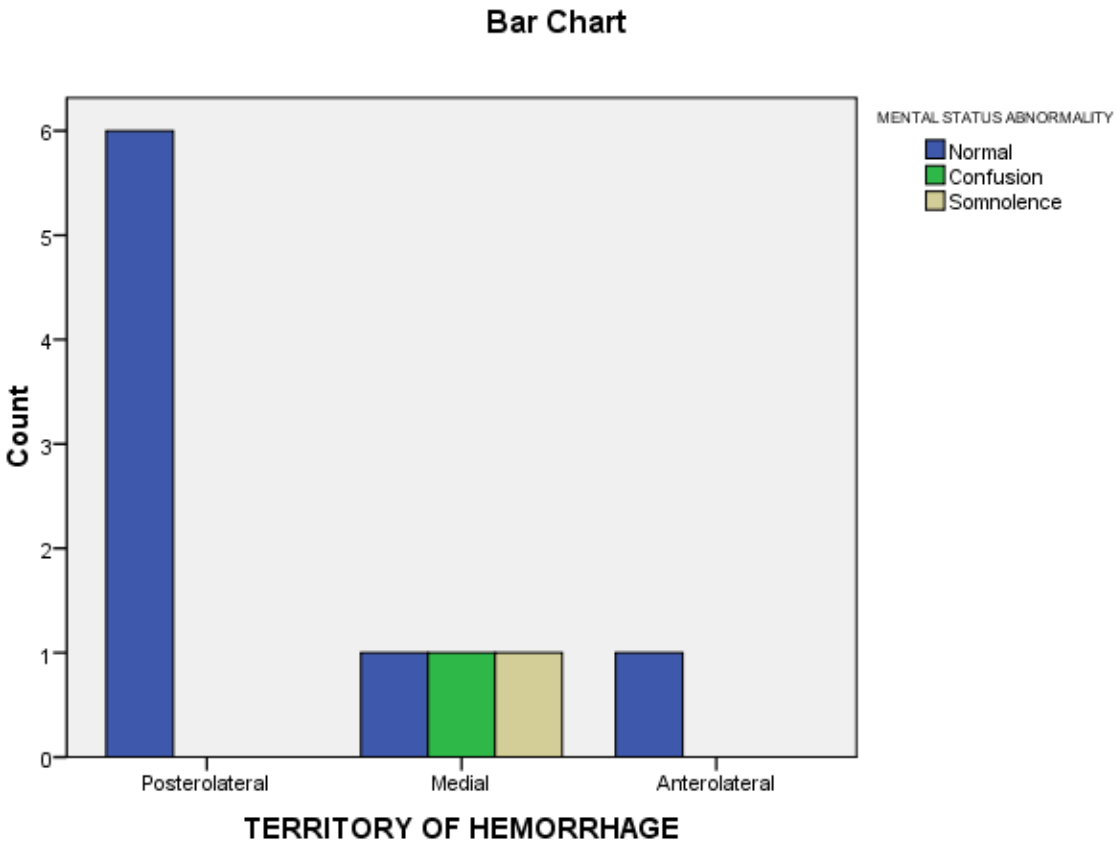
Behavioural Abnormality

TERRITORY OF HEMORRHAGE	BEHAVIOURAL ABNORMALITY			
	Normal	Frontal dysfunction	Dysphasia	Amnesia
Anterolateral	0	1	0	0
Medial	1	0	1	1
Posterolateral	6	0	0	0
Dorsal	0	0	0	0
Total	7	1	1	1



Mental Status Abnormality

TERRITORY OF HEMORRHAGE	MENTAL STATUS ABNORMALITY		
	Normal	Confusion	Somnolence
Anterolateral	1	0	0
Medial	1	1	1
Posterolateral	6	0	0
Dorsal	0	0	0
Total	8	1	1



DISCUSSION

In our study of 30 patients of thalamic vascular syndromes , 27(70%) were male patients , 9 (30%) were females . It is similar to other studies where male predominance was seen like suleyman et al where also nearly 2/3 males seen. In our study of 30 patients , the presenting age varies between 30 years to 78 yrs.

AGE GROUP PATTERN

AGE GROUP	MALE	FEMALE	TOTAL
30- 40	**1**	**0**	**1**
40-50	**3**	**3**	**6**
50-60	**6**	**1**	**7**
60-70	**7**	**4**	**11**
70-80	**4**	**1**	**5**
TOTAL	21	9	30

The mean age of distribution for 30 pts is 57.9 in our study.In study by suleyman et al median age is 62 yrsand in Santosh et al it it is 48.6. In other studies the median age varies between 57 to 63 yrs.

In our study of 30 patients, the stroke in younger pts (< 45 yrs) occurred in 6 (20%) pts. ,which varies between 10- 20% in other studies.

The gender distribution in younger and older patients showed a male predominance unlike the Lausanne stroke registry, in which women predominance seen.

In our study, cigarette smoking hypertension, hypercholesterolemia, diabetes mellitus, , coronary artery disease and atrial fibrillation were the most common risk factors for the ischemic thalamic lesions in order of decreasing frequency. Other rare risk factors like migraine, peripheral artery disease, and vasculitis are not seen

DIABETES MELLITUS

Diabetes was seen in 7 (23.3%) pts in our study, out of which 6 were males and 1 is female.

In study by sulaeiman et al diabetes was seen in 27% of patients.

HYPERTENSION

Hypertension was seen in 12 (40%) pts. 10 males and 2 females. It is seen in 50% of thalamic hemorrhage pts.

In other studies it varies from 41 to 69%.

CIGARETTE SMOKING

It is seen in 13(43.3%) of pts . all of them are males . 10 had infarct 3had thalamic hemorrhage.

CAHD

It is seen in 6(20%) pts. 5 are known pts .

1 pt had atrial fibrillation causing cardioembolic stroke.

DYSLIPIDEMIA

It is seen in 12 (40%) pts . About 60% of them are diabetic or hypertensive or both.

In other studies it is 32%

CT SCAN BRAIN

CT brain was done in all 30 patients. 19 (63.3%) had abnormal scan.All 10 thalamic hemorrhage had abnormal scan. 9 infarct pts had abnormal scan initially.Normal initial CT scan was seen in 11 pts with infarct.

NATURE OF STROKE

Out of 30 pts , 20(66.7%) had infarction . 10(33.3%) had thalamic hemorrhage. Other studies showed a 20 to 33% thalamic hemorrhage in thalamic stroke.

Pattern of infarct

Out of 20 thalamic infarct patients 11(55.5%) had Thalamogeniculate infarctions, 5(25.5%)had .paramedian infarct, 2(10%)had polar infarct ,1(5%) each had posterior choroid and bilateral paramedian infarct

Thalamogeniculate infarction was seen in 55.5% in our study compared to

45 to 55% in other studies.Which is 51 % in suleyman et al.

25% had paramedianinfarct,itis 30 % in suleyman et al.

, 2(10%)had polar infarct ,1(5%) each had posterior choroid and bilateral paramedian infarct which is similar to other studies.

PATTERN OF HEMORRHAGE

Out of 10 hemorrhagic thalamic strokes, 6 (60%) had posterolateral, 3(30%) had medial, 1 (10%)hadanteolateral haemorrhages.

The commonest site isposterolateral which is similar to

Caplan andwang et al.

CLINICO – RADIOLOGICAL CORRELATION

Pattern of weakness

Motor weakness involving UL& LL occurred in 5% in paramedian infarct, which is 15% in the study by emrekumral et al

Weakness in face , UL & LL seen in 20% in Thalamogeniculate infarctions, 5% in paramedian infarct, which is similar to the quoted study.

SENSORY

Hemisensory loss seen in 45% of Thalamogeniculate infarctions, 10% of paramedian infarct, 5% polar infarct .It is 60 to 87% in suleyman et al.

Dejerine –Roussy syndrome is seen in 10% of pts. Which is 17% in suleyman et al

ATAXIA

It is seen in 3Thalamogeniculate infarctions which is similar to suleyman et al

, 1paramedian infarct which is lesser than suleyman et al and in

2 polar infarct ,1 posterior choroid and infarct.

OCULAR DEFICIT

Vertical gaze palsy Is seen in 1 paramedian infarct, posterior choroid and bilateral paramedian infarct which is seen in 65% in Bogousslavsky et al

VISUAL FIELD DEFECT

Defects like quadrantanopia is seen in 1 posterior choroid which is similar to kumral et al

BEHAVIOURAL CHANGES

1 Patient had frontal dysfunction and dysphasia in polar hemorrhage.2 patients had amnesia in thalamogeniculateinfarct.This is similar to study by kumral et al.

MENTAL CHANGES

Confusion is seen in 1 each in Thalamogeniculate infarction. paramedian infarct, and polar infarct and somnolence in 2 pts with paramedian infarct,

It is similar to the study bysuleyman et al

CLINICO-RADIOLOGICAL CORRELATON IN THALAMIC HEMORRHAGE

Motor weakness

In our study face and upper limb weakness is present in one patient of poster lateralhaemorrhage, upper limb and lower limb weakness is seen in each one patient in anterolateral,medial,posterolateralhemorrhages.This is similar to the study by suleyman et al.

Sensory deficit

Hemi sensory loss was seen in 3 patients in posterolateral,2 patients in medial and 1 patient in anterolateral haemorrhage. In study by kumral et al hemi sensory loss was seen 52%.

Ataxia

Ataxia was seen in 1 patient with anterolateral and medial hemorrhages.Ataxia due to posterolateral haemorrhage which was seen in kumral et al study, was not seen in our study.

Ocular abnormalities

Skew deviation was seen in 1 patient with medial haemorrhage. Inkumral et al study gaze palsy was seen in 67% ofposterolateralhaemorrhage ,was not seen in our study.

Visual field deficit

It was seen in 1 patient with anterolateral haemorrhage, which is 3% in kumral et al study.

Behavioural disturbances

Frontal dysfunction is seen in 2 patients with medial and 1 patient with anterolateral haemorrhage.

Dysphasia was seen in 1 patient with medial haemorrhage, which was seen 30% of posterolateral and medial haemorrhages in kumral et al study.

Mental abnormalities

Confusion and somnolence is seen in 2 patients of medial haemorrhages.

Somnolence was seen in 30% of posterolateral,13% of medial and anterolateral and 21% of dorsal haemorrhage.

In our study, the most frequentcauses of isolated thalamic ischemic strokes were small artery disease inthree fourths of the patients, and cardiogenic brain embolismin one patient. Largeartery disease was not seen in our study than in a previousstudy in which large artery disease was found in(18%).

In our study, the frequency of infarction in the territoryof thethalamogeniculate artery was higher than in theother territories of thalamic arteries, as reported previously.

CONCLUSION

1) This study shows male preponderance in the acute thalamic stroke.

2) In this study commonest age group affected is >60 years (53.5%) followed by 40 to 60 years (43%followed by <40 years (3.3%).

3) In this study there is predominance of thalamic infarction(70%) over haemorrhage(30%)

4) In this study, most common risk factors seen are smoking (49%), hytertension &dyslipidemia (40%) , Diabetes Mellitus (23%) Coronary Heart Disease (20%) & Atrial Fibrilation (3 %).

5) Rare risk factors like vasculitis and migraine are not seen.

6) Only one patient had cardioembolic stroke due to non valvular Atrial Fibrilation

7) The commonest territory involved is thalamogeniculate in thalamic infarcts.

8) The commonest clinical presentation is hemisensory loss due to involvement of thalamogeniculate territory

9) The commonest territory involved is posterolateral in thalamic Haemorrhage.

10) The commonest presentation in posterolateral haemorrhage is hemisensory loss and hemiparesis.

11) Posterior choroidal territory is least commonly affected in thalamic infarct.

12) Dorsal area is least commonly affected in thalamic haemorrhage.

commonest area seen in altered sensorium is paramedian area.

THIS STUDY ENABLES US TO UNDERSTAND THE THALAMO CORTICAL NETWORK BETTER.

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PROFORMA

Name :

Serial No :

Age :

IP No. :

Sex :

Address :

DOA :

DOD :

Phone No. :

Diagnosis :

History:

Complaints :

HOPI :

Past History :

HT :

DM :

CAHD :

CVA :

Others :

Clinical Findings:

General Examination:

CNS :

Higher Mental Functions:

Motor System :

Sensory System :

Cranial Nerves :

Cerebellar System :

Autonomic System :

Meningeal Signs :

Investigations : **Basic Investigations**

Blood :

Others :

Radiology:

1. CT Brain and CT angiogram in selected cases
2. MRI Brain with MRA and MRV and contrast study is selected cases

Clinical Outcome:

நோயாளியின் ஒப்புதல் படிவம்

மூளை நரம்பு முடிச்சு இரத்த குழாய் நோய்குறியில் மருத்துவ நோய்சார்ந்த மற்றும் கதிரியக்க ஒற்றுமை ஆய்வு

ஆய்வு நிலையம் : **நரம்பியல் துறை, இராஜீவ்காந்தி அரசு பொது மருத்துவமனை, சென்னை மருத்துவக் கல்லூரி, சென்னை- 600 003.**

பங்கு பெறுவரின் பெயர் :

பங்கு பெறுவரின் எண் :

பாலினம் :

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக்கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கின்றேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிகிறேன். ☐

நேரம் :

நாள் :

இடம் :

கலந்து கொள்பவரின் கைரேகை/
கையொப்பம்
பெயர்

ஆய்வாளரின் கையொப்பம் :

ஆய்வாளரின் பெயர் :

MASTER CHART

Number	Sex	Age	Diabetes	Hypertension	Dyslipidemia	Coronary	Fibrillation	Smoking	CT	Territory	Motor	Sensory	Ataxia	Ocular	Visual	Behavioral	Mental	Stroke typ	H-TERRITORY
1	1	30	2	2	2	2	2	1	1	5	1	2	2	2	2	1	3	1	0
2	1	48	2	1	2	2	2	1	2	1	1	2	2	1	2	1	1	1	0
3	2	73	2	2	1	2	2	2	2	1	4	2	2	1	2	1	1	1	0
4	1	75	2	2	2	2	2	2	1	1	4	2	1	3	2	1	1	1	0
5	1	67	1	1	2	2	2	2	2	1	1	2	2	1	2	4	2	1	0
6	2	51	2	2	2	2	2	2	1	1	1	2	1	1	2	1	1	1	0
7	1	58	2	2	2	2	2	1	2	1	1	4	2	1	2	1	1	1	0
8	1	66	1	1	2	2	2	2	1	1	1	2	2	1	2	1	1	1	0
9	2	68	2	2	1	1	2	2	2	1	4	2	2	1	2	4	1	1	0
10	1	62	2	1	2	1	2	1	1	1	1	4	1	1	2	1	1	1	0
11	1	40	1	2	2	2	2	1	1	1	4	2	2	1	2	1	1	1	0
12	1	64	2	1	2	2	2	1	2	1	1	2	2	1	2	1	1	1	0
13	1	78	1	1	1	2	2	2	1	0	3	2	2	1	2	1	1	2	1
14	1	54	2	2	1	2	2	1	1	0	4	3	2	1	2	1	1	2	1
15	2	62	2	1	1	2	2	2	1	0	1	2	2	1	2	1	1	2	1
16	1	50	2	1	2	2	2	2	1	0	1	2	2	1	2	1	1	2	1
17	2	40	2	2	1	2	2	2	1	0	2	1	2	1	2	1	1	2	1
18	1	70	2	2	2	2	2	1	1	0	4	1	2	1	2	1	1	2	1
19	1	65	1	1	1	2	2	1	2	2	1	1	2	2	2	1	3	1	0
20	2	64	2	2	1	2	2	2	1	2	1	1	2	1	2	1	3	1	0
21	1	57	2	1	2	1	1	1	2	2	1	2	2	3	2	5	2	1	0
22	1	72	2	2	2	2	2	2	2	2	4	2	1	1	2	5	1	1	0
23	1	55	1	2	1	2	2	1	1	2	2	1	2	1	2	1	1	1	0
24	2	63	2	2	2	1	2	2	1	3	1	1	1	1	1	3	1	1	0
25	2	40	2	2	1	2	2	2	2	3	1	1	1	1	1	4	2	1	0
26	1	62	1	2	2	2	2	1	2	4	1	2	1	1	2	1	1	1	0
27	2	40	2	1	1	1	2	2	1	0	1	1	2	1	2	1	3	1	2
28	1	62	2	2	1	2	2	2	1	0	2	2	2	3	2	4	2	1	2
29	1	43	2	1	2	1	2	2	1	0	4	2	1	1	2	5	1	1	2
30	1	58	2	2	2	2	2	1	1	0	2	2	1	1	1	3	1	2	3

Sex	MALE	1
	FEMALE	2
Diabetes	PRESENT	1
Hypertension	ABSENT	2
Dyslipidemia		
Coronary		
Fibrillation		
Smoking		
CT		
Territory	THALAMOGENICULATE	1
	UNILATERAL PARAMEDIAN	2
	POLAR	3
	POSTERIOR CHOROIDAL	4
	BILATERAL PARAMEDIAN	5
Motor	NORMAL	1
	UL & LL	2
	FACE & UL	3
	FACE, UL & LL	4
Sensory	NORMAL	1
	Hemi sensory	2
	Cheiro-oral	3
	De´jerine-Roussy syndrome	4
Ataxia	PRESENT	1
	ABSENT	2
Ocular	normal	1
	Vertical gaze palsy	2
	Skew deviation	3
	Pseudo-VI	4
	III CRANIAL NERVE PALSY	5
Visual	PRESENT	1
	ABSENT	2
		Mental
Behavioral	normal	1
	Dementia	2
	Frontal dysfunction	3
	Dysphasia	4
	amnesia	5
	Visuospatial neglect	6
Mental	normal	1
	CONFUSION	2
	SOMNOLENCE	3
Stroke type	INFARCTION	1
	HEMORRHAGE	2
HEMORRHAGE TERRITORY	POSTEROLATERAL	1
	MEDIAL	2
	ANTEROLATERAL	3
	DORSAL	4

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr.D.Sekar
PG in D.M.Neurology,
Madras Medical College & Rajiv Gandhi GGH,
Chennai -3

Dear Dr.D.Sekar,

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled " Clinico-Radiological correlation in Thalamic vascular syndromes" No.36112012.

The following members of Ethics Committee were present in the meeting held on 01.11.2012 conducted at Madras Medical College, Chennai -3.

1. Prof. R. Nandhini MD Director, Instt. of Pharmacology ,MMC, Ch-3	-- Member Secretary
2. Prof. Reghu MD Director , Inst. Of Internal Medicine, MMC, Ch-3	-- Member
3. Prof. Shyamraj MD Director i/c , Instt. of Biochemistry , MMC, Ch-3	-- Member
4. Prof. P. Karkuzhali. MD Prof., Instt. of Pathology, MMC, Ch-3	-- Member
5. Prof. G.Muralidharan MS Prof of Surgery, MMC, Ch-3	-- Member
6. Thiru. S. Govindsamy. BA,BL	-- Lawyer

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nandini 23/11/12
Member Secretary, Ethics Committee